ANALYTICAL AND DIFFERENTIAL DIAGNOSIS OF NERVOUS DISEASES

HENRY HUN, M.D.

AN ATLAS

OF THE

DIFFERENTIAL DIAGNOSIS

OF THE

DISEASES OF THE NERVOUS SYSTEM

(WITH A PHYSIOLOGICAL INTRODUCTION)

ANALYTICAL AND SEMEIOLOGICAL NEUROLOGICAL CHARTS

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To
Thomas Hun
a loving father
a learned physician
a man of wisdom and wit
this book is dedicated
in most grateful remembrance.

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PREFACE

The diagnosis of diseases of the nervous system is generally regarded by medical students as one of the most difficult subjects in their course of study. It is so difficult that many students become discouraged and after a few attempts make no strong, continued effort to master it and, perhaps in consequence, physicians generally are weaker in this than in other phases of their work. In the hope of making this task less difficult for both physicians and students this book has been written. If the student can be taught to make the diagnosis of these diseases with comparative ease, it may happen that he will be led to undertake those further studies in the finer anatomy and physiology of the nervous system, which are essential for a full understanding of this difficult but fascinating department of medicine.

A careful physical examination and history of the case, as complete as can be obtained, are, of course, the essential basis of every diagnosis; but the commonly employed method of comparing the combination of symptoms thus obtained in any case with the various syndromes characteristic of the different diseases until a similar combination can be found, is not altogether satisfactory. More scientific and instructive are the analysis of each important symptom, its explanation in the normal or abnormal activity of the nervous system and the consequent ascertaining of the disease which must cause it under the circumstances (the other symptoms) existing in any individual case which may present itself.

In spite of its apparent complexity, the diagnosis of nervous diseases lends itself better than that of the diseases of most of the other organs to exact pathological analysis. Just as a chemist in analyzing a substance of unknown composition by a series of appropriate tests eliminates from consideration one group of chemical bodies after another until he finally discovers its class and name, so the neurologist subjects a patient to one test after another in definite sequence. As the result of each test he throws out of consideration one or more groups of diseases and assures himself that he has to do with a disease belonging to another definite group. With each successive test the number of diseases constituting a group becomes less, until finally one definite individual disease stands revealed among the few most closely related to it and can be absolutely identified by a comparison of the remaining symptoms characteristic of each, which are given in the final abstracts. This analytical method is used, I think, by most teachers of neurology in demonstrating cases of diseases before their classes of students. It is the crystallization of this teaching into the tabular form which this book attempts to present.

In using this book for diagnostic purposes it is important that the "Introduction to the Diagnostic Charts" on page 119 should be carefully studied. By means of these charts it is possible to diagnosticate easily and rapidly almost any

disease of the nervous system and to localize the lesion, when any lesion exists. If the examiner makes a mistake at any point, the next step in the process or the abstract of the other symptoms of disease will probably show him that he is in error and that it is necessary for him to retrace his steps.

For the sake of completeness certain trophic diseases are included, which, although causing a number of functional disturbances in the nervous system, are not really nervous diseases.

As might naturally be expected, the same disease, in so far as it presents many symptoms, appears a number of times in the different charts and even in the same chart; so that, in order to get a more complete idea of its symptomatology, it is essential that the different abstracts of it should all be read. To facilitate this, cross references by numbers within brackets are placed in the text.

Many diagnostic and technical terms are used which may not be familiar to the student; therefore these terms are classified, defined and their significance stated, as far as it is known to the author, in a series of semeiological charts preceding the diagnostic ones. Cross reference to these terms also is facilitated by the numbers within the brackets. A very complete index serves this same purpose.

The peculiar characteristic of this book on diagnosis is that it gives to the student or physician a key by which, in a comparatively easy manner from one or more important symptoms, he can arrive at a diagnosis. It also has the advantage that it divides the diseases into groups, the members of which have a definite relationship with each other; so that in the process of using the charts the student is constantly catching glimpses of the natural relationships between the different diseases of the nervous system. Although the symptoms of different diseases have often been contrasted in tables of parallel columns, in no other book, known to the author, has the subject been presented as it is here, and this must be his excuse for publishing it and for any defects which it may show, as there was no model which could be followed in preparing it.

In the preparation of this book the author has received valuable suggestions and aid from several friends and especially from Drs. Archambault, Dawes, Hawn, Mosher, Streeter and Viets. To these, his present friends and former students and assistants, he gratefully acknowledges his indebtedness and returns his thanks.

It is very gratifying to the author that the first two editions of five thousand copies have been exhausted, giving him an opportunity of making a complete revision of the text, and of making a few additions both to the semeiological and to the diagnostic charts, which seem to him to add much to the value of the work.

The criticisms of Dr. Mosher and his untiring aid in putting the three editions through the press are large factors in whatsoever success the book may have.

HENRY HUN.

Albany, N. Y.,
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PART I

SEMEIOLOGY

THE EXAMINATION OF PATIENTS

AND A

PHYSIOLOGICAL AND PATHOLOGICAL ANALYSIS

OF THE

RESULTS OBTAINED FROM SUCH EXAMINATION

AN ANALYSIS OF THE
SUBJECTIVE AND OBJECTIVE SYMPTOMS OF DISEASE

Introduction to the Semeiological Charts

A Review of the Physiology of the Nervous System

(The bracketed numbers refer to the numbers in the body of the book)

The diagnosis of nervous diseases, if it is to be at all satisfactory and accurate, must be based on anatomy and physiology. It might seem strange to mention physiology in connection with such a schematic tabulation of diagnosis as this book presents. All of these charts, however, are constructed strictly on a physiological basis and without such basis they could not have been made or could not be successfully used. The diminution, exaggeration, or perversion of the different physiological functions of the nervous system constitutes the scheme of each chart and is the key for the diagnosis of the separate diseases which are their clinical expression. The practitioner is supposed to have some acquaintance with these subjects, and the curriculum of the medical college is so arranged that the student is taught them before he commences clinical work. It seems, however, desirable to make here an attempt to present a very brief, but comprehensive, outline of the physiology of the nervous system, including some statements as to its anatomy, which latter can be supplemented by an inspection of the plates at the end of the book.

The human being is a unit in the universe which contains him, of which he is a part and with which he is in constant relationship. Life consists in a constant reaction of the organism to the forces of nature which act upon it from all sides. These reactions are called "physiological activities," and when they become disordered from any cause they are called "pathological activities," or symptoms of disease; when they are altogether absent the organism is dead. The nervous system is the most important seat of these reactions. Of the numerous forces and forms of energy in the world only a portion can be perceived by man. It is certain that some animals perceive things imperceptible to him.

The various forms of energy in nature cannot act directly upon the nervous system to produce sensory impulses; but intermediate organs, "end-organs," or "receptors," are necessary to transmute into nervous energy, the external energy (sun's rays, etc.) which comes from the external world. There may be some question whether the external energy passes through the nervous system to the muscles and from them passes out again to the external world; or whether the external energy (light, etc.), in its action upon the end-organ, liberates the potential energy stored in the end-organ, just as a spark liberates the potential energy stored in gunpowder. The latter assumption seems to be the true one, because the energy imparted to the animal by the stimulus is much less than the consequent energy manifested by the animal. Hence the conclusion, that in virtue of chemical changes the potential energy stored in the end-organ becomes active; the exciting cause of this being the external force or foreign irritation.

There are doubtless many forms of energy in the world which cannot be perceived by man because he possesses no suitable end-organs to bring about this transmutation. Sometimes this can be accomplished by adding to the end-organs some mechanical contrivance suitable to bring about this transmutation; as, for instance, the fluoroscopic screen for X-rays. The universal ether is doubtless in vibration far beyond the limits of about four hundred million million per second, which constitute the color red and about seven hundred and sixty million million, which constitute violet; and, indeed, we have reason to believe that the ultra-violet rays have some effect upon the human body, but beyond the above limits the vibration of the ether is neither reacted to by our eye nor recognizable by our brain. The sensory apparatus is not perfect. Some time is required for the excitation caused by the external stimulus to pass through the end-organ and the nerve fibers to the cerebral cortex and to excite the latter to action (the initial lag) and the resulting sensation persists an even longer time after the external stimulus has ceased (the terminal lag). Things moving very rapidly, as for instance the spokes of a rapidly moving wheel, cannot be

seen individually, and a rapidly recurring series of the same sound blends into a musical tone. But, however limited and imperfect the sensory apparatus may be, the end-organs, each of which is specific and can respond to one definite form of energy and to that one only, whether on the surface or in the interior of the body, are starting points from which all nervous and mental activity, even the highest, springs. The fundamental function of the end-organ and hence the primary activity of the nervous system is, thus, "receptivity."

The fundamental, anatomical element, or unit, of the nervous tissue is the "neuron"(461-4): a nucleated cell with many processes projecting from it, some short and branching (dendrons); one (rarely two or more), often extending a long distance, usually becomes the axon of a medullated nerve fiber, and frequently gives off a few collateral branches.

Both axons and dendrons are composed of delicate fibrillae which pass directly, without interruption, through the cell body from the tip of a dendron to the tip of the axon. Of these neurons, varying in form and size and supported by the delicate framework of the neuroglia, the entire nervous system is composed.

The fundamental physiological characteristics of the nervous tissue are excitability and transmission: the power of receiving an excitation and transmitting it from one end of the neuron to the other, and even to other neurons with which the first has anatomical and physiological relationship, or contact. By its dendrons the nerve cell receives nervous impulses and by its axon transmits these impulses. The transmission of energy from one neuron to another at their *synapse,* or point of contact, depends, probably, upon differences in the tension of this energy in the two neurons. Later observations, however, seem to prove that, at the termination of the axon, its fibrillae pass directly into the dendron of another neuron, at least in some cases. A neuron is merely a conducting channel. It originates nothing. It merely transmits.

The rapidity of conduction of the nervous impulse along a nerve fiber is approximately four hundred feet per second normally, but varies with the temperature and other artificial conditions. The nervous conduction seems to be a wave of chemical decomposition associated with a local electrical current. The transmission of an impulse from one neuron to another is relatively slow.

The Centripetal Neurons

Cutaneous Sensibility. The surface of the body and the cavities connected with it contain organs: the terminal organs of sense (the sensory "end-organs"), which bring the body into connection with some, but probably far from all, of the forces of nature and these end-organs are so constructed as to transmute physical forces (light in the eye, sound in the ear, heat, cold, touch, pressure and pain in the skin, etc.,) into nervous excitations in the terminal filaments of the peripheral nerves. The skin contains many of these *isolated* terminal sense organs and, therefore, sensibility is not spread uniformly over the skin, as it appears to be, but is really located in individual points lying close together, but each distinct. From these points of greatest sensibility its acuteness diminishes concentrically. In every square centimeter of skin there are, on the average, 12 to 13 points for cold, 0 to 3 for heat, and 24 for tactile impressions; although these figures vary very greatly for different parts of the skin, the points being most numerous on the finger tips and fewest on the back. Points for tactile impressions vary from 7 to 300 per square centimeter. There are also distinct points for painful sensibility. These points for painful sensibility are much more numerous, but far less sensitive and delicate, than are those for tactile sensibility. The nerve fibers supplying these points of painful sensibility have no end-organs, but end as fine free filaments. Where the skin is stretched over bone (*e.g.*, the malleoli) tactile sensibility is less distinct, and it is more distinct where the skin is hairy; a point for tactile sensibility being situated at the base of most, if not of all, hairs. There may be a delay in the conduction of painful impressions through either the neuron or the synapse; so that with repeated pin-pricks the pain becomes more acute (summation of impulses). After section, or injury, of a nerve, the anesthesia and analgesia are never so extensive as is the area of distribution of the nerve, and sensibility often returns before the regeneration of the nerve has completely taken place. This is partly due to peripheral anastomoses with terminal filaments of adjacent nerves and partly, perhaps, to sensory fibers in the motor nerves (recurrent sensibility.)

Head and his colleagues, after much experimental work on the subject, arrived at the following conclusions:

There are in the peripheral areas three kinds of sensibility, due to there being three different kinds of nerve fibers supplied to each area:

1st. *Deep Sensibility*: Muscle sense, joint sense, pressure, pain, and vibration sense. This sensibility is conveyed by sensory nerve fibers, more or less deeply situated beneath the skin, and usually running with the motor nerves. When the nerves of the tendons are cut, these forms of sensibility are lost, but not on section of the sensory nerves of the skin.

2nd. *Epicritic Sensibility*: Tactile sensibility for slight impressions, form and space sense, sense of moderate, not extreme, temperatures, (22° to 40° C.) and the precise localization of pain and temperature sense. This area of sensibility is very constant for each individual nerve. The restitution of this form of sensibility is very slow and is not complete until several years after the injury to the nerve.

3d. *Protopathic Sensibility*: Pain and sense of extremes of temperature (below 22° or above 40° C.). Disturbances of this form of sensibility are accompanied by paresthesiae and a false localization. This form of sensiblity is best tested on the periphery of the affected area, where the anesthesia is not complete, or over the whole area while the regeneration is taking place. The restitution of this form of sensibility is relatively rapid (7 to 10 weeks).

These researches of Head are of great interest and value and have attracted much attention and discussion: but they are not, in their entirety, accepted by all neurologists and more recent investigations seem to show that after section or injury of a nerve all forms of sensation are practically equally impaired and in cases of recovery all return with equal rapidity; so that testing for one form usually suffices for all. The testing of sensibility is very difficult, so much depends upon the patient's intelligence, power and quickness of perception and freedom from suggestion.

Sensory impulses of all kinds are carried to the central nervous organs by the sensory nerves. Of these, the spinal nerves enter the cord (mainly) through their cell bodies in the spinal ganglia and through the posterior nerve roots (Fig. 26); while the fibers of the trigeminal, the great cranial nerve supplying sensation to the face, after passing through their cell bodies in the Gasserian ganglion, enter the pons (Fig. 19). The fibers from the posterior nerve roots, on entering the spinal cord, are sorted according to their physiological function into three great groups (Fig. 26). One group which conveys most of the tactile, and a part of the pressure and muscle-joint sense impressions, ascends mainly, without decussating, in the posterior columns to the nuclei of the columns of Goll and Burdach, and thence is continued by a new set of neurons (the internal arcuate fibers), which decussate and pass through the median lemniscus (fillet) (Figs. 20-3) to the optic thalamus; whence it is continued, also by another set of neurons (relays), to the parietal cortex. These fibers convey impulses essential to the production of association reflexes. The second group, which conveys impulses essential to the maintenance of equilibrium, passes to the cells of the columns of Clarke and thence, mainly without decussating, through the direct cerebellar (posterior, spino-cerebellar or Flechsig's) tract in the outer part of the lateral column and through the restiform body to the vermis or middle lobe of the cerebellum. The third group, which conveys all the temperature and painful impressions, passes through cells in the posterior horn, decussates in the central gray matter of the cord and passes upward through the spino-thalamic tracts and lateral portion of the formatio recticularis, joining in part the median lemniscus, to the optic thalamus and thence to the parietal cortex, and in part coursing backwards to the cerebellum. The fibers of the first and third group have thus a double ending, one in the optic thalamus and one in the cortex.

A destructive lesion, either in the terminal end-organ or at any point of these sensory tracts or neurons, causes a corresponding paralysis of sensation (anesthesia); while a slight, irritative lesion may cause hyperesthesia, paresthesiae or pain in the distribution of the nerve.

In addition to the anesthesia, which occurs in organic disease of the nervous system, there is an anesthesia which occurs in hysteria; hysterical anesthesia (415). This hysterical anesthesia occasionally, but rarely, involves the organs of special sense. It more commonly involves cutaneous sensibility and then the anesthesia is not limited to the distribution either of a peripheral nerve or of a nerve root. It may instantaneously disappear. It may recur in the same place, or in some other locality. It does not prevent the use of the part in performing acts in which sensibility is essential. Upon excitation of the hysterical anesthetic parts vascular reflexes occur, which is never the case in anesthesia due to organic disease, peripheral or spinal. It is evident that this hysterical anesthesia is a purely mental phenomenon: an imagined idea or delusion. It is due to suggestion; a "fixed idea" is so dominant that the sensation of pain or touch, which should normally result, does not enter consciousness (see later). The excitation causing the dominant fixed idea inhibits other cortical activity.

Pain (374) is an unpleasant sensation which never occurs in health, but only when the body is injured, either mechanically or chemically. It is a signal or warning that the body needs protection. Its intensity depends not only upon the intensity of the mechanical or chemical irritation, but also upon the condition (inflammatory, etc.) of the peripheral nerves or of the cerebral cortex. It is more intense when cerebral cellular activities produce the concepts of fear and apprehension and the condition of "active attention" (see later), which makes the painful sensation more acute. An unexpected wound is less painful than an anticipated one. and a wound is often not at all painful when the cortical activity is greatly excited by some other cause (a battle for instance.) Pain is often associated with the allied perceptions called "paresthesiae" (375), which at times accompany, and at times follow, the pain and which are usually of central origin and are due to irritation of the sensory fibers at some part of their course through the central nervous organs.

The Kinesthetic Sense (43). The muscles, tendons and articular surfaces all contain end-organs which send impulses to the central nervous organs whenever the muscles contract or the joints move and cause sensations from which the position of the limb in space can be accurately located, even when the eyes are closed or the patient is blind. These sensations are caused by the movement of the part and have much to do with regulating and inciting its further movements. The kinesthetic sense is of the greatest importance in all act-

ions and more especially in the automatic and habitual acts, (walking, swimming, talking, singing, etc.). The pathways for the nerve fibers conducting "muscle-sense," as it is often called, and which have their origin in the muscle spindles, etc., enter the spinal cord through the posterior nerve roots, mainly pass forwards to the columns of Lockhart Clarke and thence directly outwards to the direct cerebellar tract (posterior spino-cerebellar tract) and through it to the cerebellum, and in part through the posterior columns to the cerebrum. The corresponding fibers in the cranial nerves enter the pons and medulla.

The Equilibrium Sense. The three semi-circular canals, each lying in a plane at right angles to the other two, constitute the most important organ for the equilibration of the body. In every movement of the head the endolymph in the canal in the plane in which the head is moved is set in motion and moves also the hair-cells in the canal. The stimulus arising from these irritated hair-cells passes through the vestibular branch of the acoustic nerve to Deiter's nucleus and its neighborhood and thence to the vermis of the cerebellum. The vestibular nerve has no direct connection with the cerebral cortex and hence its activity is entirely unconscious.

Gustatory Sensation. (Charts VIa and XIVe). The mucous membrane of the mouth (in addition to the terminal organs for tactile, pressure, thermic, painful, etc., impressions) contains also the terminal organs of the nerves of taste: the taste-buds or bulbs, so called from their form, embedded in the epithelium of the mucous membrane of the mouth, especially in the tip and edges of the tongue. Excitation of these taste-bulbs gives rise to four distinct gustatory sensations: sweet, acid, salty and bitter, to which may, perhaps, be added alkaline and metallic. Many so-called tastes are really a combination of gustatory and olfactory sensations. The nerve fibers arising from the taste-bulbs on the posterior portion of the tongue pass by the glosso-pharyngeus nerve in a direct manner through the petrous ganglion to their nucleus in the medulla, whence they ascend with the other sensory fibers of the lemniscus to the optic thalamus, and thence to the cerebral cortex, posterior to the olfactory area, in the gyrus hippocampi, (Fig. 16); while the nerve fibers arising from the taste bulbs on the anterior portion of the tongue pass at first in the chorda tympani nerve and run with it through the Fallopian canal to the geniculate ganglion. Here the fibers divide; a part continuing alongside the facial nerve and forming the nervus intermedius, which runs to a nucleus in the medulla, close to the glosso-pharyngeal nucleus; while the rest of the fibers run through the petrosal nerve and join the fifth nerve and pass to the Gasserian ganglion (Fig. 36), and thence to the cerebral cortex (Fig. 16). A destructive lesion at any part of this course will cause unilateral loss of taste (ageusia). When the lesion is in the Fallopian canal, the ageusia may be associated with facial paralysis on the same side.

Olfactory Sensation. (Charts VIa and XIVe). The mucous membrane of the nose, analogous to that of the mouth, in addition to the end-organs for tactile, thermic, pressure and painful impressions, contains also the end-organs of the nerves of smell. The nerves terminating in these organs pass upward through the cribriform plate to the olfactory bulb, and thence backward through the olfactory tract; some to the anterior perforated space and sub-thalamic region (for the olfactory reflexes) and others to the cortical centers for smell in the cornu Ammonis (Fig. 16). These fibers do not pass through the internal capsule as do the fibers from all the other organs of sense, with the doubtful exception of the gustatory fibers.

Hearing. (Charts VIa and XIVe). The terminal organ for hearing is the organ of Corti in the cochlea within the petrous portion of the temporal bone. In this organ there is a long series of vibratory structures of unequal length supported upon the basilar membrane (membrana spiralis); so that among them can be found a representative for every possible tone (produced by single vibrations of any rapidity between 40 and 40,000 per second) with its overtones, or harmonics. The acoustic nerve (cochlear nerve) arising in this organ passes to the ventral and dorsal acoustic nucleus in the pons (Fig. 19). From this nucleus fibers pass upward, some decussating and some not, through the lateral fillet (lemniscus lateralis) to the posterior corpora quadrigemina and the median geniculate body ofthe thalamus and thence through the sublenticular region of the internal capsule, posterior to the fibers for cutaneous sensibility, and reach the cortical auditory area in the anterior transverse temporal convolution immediately below and forming the lower margin of the Sylvian fissure in each hemisphere. A destructive lesion of one acoustic nerve will cause unilateral deafness on the same side, but a lesion of the tract connecting the subcortical with the cortical centers, since this tract contains both crossed and uncrossed fibers, will not cause any deafness The cortical representation of the auditory nerve is bilateral. Deafness results only from a *bilateral* central lesion affecting the corpora quadrigemina or the subcortical tracts described above. Even destruction of the acoustic cortical center in both hemispheres does not seem always to cause complete deafness.

Sight (Charts VIa and XIV, b, c, d). The terminal end-organs for sight consist of the rods and cones in the retina. The rods seem to be concerned with seeing in dim, the cones in bright, light. They vary in relative numbers in different animals, according as the latter roam by night or by day. They also vary in man according to their situation. Only cones are found in the fovea centralis. These gradually diminish in number towards the periphery of the retina, where only rods are found. From these structures start the terminal filaments of the optic nerves, which run backward from the eyeballs to the optic chiasm. In the optic chiasm the fibers from both maculae luteae and from the nasal half of each retina decussate; so that in the left optic tract are collected all the fibers from the left half of each retina (right visual field) and those from both maculae luteae; while in the right optic tract are collected all the fibers from the right half of each retina (left visual field) and those from both maculae luteae. The fibers of the optic tract on each side terminate in

the external geniculate body, the pulvinar and the anterior quadrigeminal body of the same side, and are thence continued through the posterior portion of the internal capsule and the fasciculus of Gratiolet to the lips of the calcarine fissure on the median surface of the occipital lobe of the same side (Figs. 16 and 37).

A destructive lesion of the optic nerve causes blindness of the corresponding eye, but a lesion of any portion of the optic tract, or geniculate body, or fasciculus of Gratiolet, or the lips of the calcarine fissure, will cause homonymous hemianopia of the field of vision of the opposite side. A lesion of the central portion of the optic chiasm will cause bitemporal hemianopia; while a lesion of the outer edge of the optic chiasm will cause nasal hemianopia of the field of vision of the opposite side.

Internal or General Sensation. In addition to these sensory impulses, which convey to the brain excitations from the special sensory organs and hence from the external world, there are others which come from the different organs, or viscera, of the body and, in case they reach the cortex, give rise to what are called internal or general sensations. Some of these internal excitations remain entirely peripheral and affect mainly the blood vessels; others reach no further than the spinal cord or ganglia at the base of the brain and incite those automatic acts which preserve the nutrition and the life of the individual, and still others reach the cerebral cortex and at times affect profoundly the processes taking place in it. These general sensations have much influence on a person's moods, thoughts and actions and especially his emotions. Hunger may entirely alter the acts of a man or beast.

Such internal or general sensations are for the most part ill-defined and ill-localized. They seem to depend upon the blood supply and the activity of the different organs and upon the state of contraction of the hollow organs; and they appear to have much to do with our feeling of comfort or discomfort, which latter may amount to great pain. The sensation of hunger seems to be caused by contraction of the empty stomach, and the various colics by contraction of the circular muscular fibers of the intestine, the ureter, or the bile duct. But the best understood of all these internal or general sensations is the composite one called "muscle-joint sense," which is mainly made up of impulses from the muscle and its tendon and the articulating surfaces and also from impulses from the skin and other tissues in the neighborhood, as these are stretched or relaxed in motions of the joint. The muscle-joint sensory conduction we have already considered under the term of the kinesthetic sense.

The centripetal neurons entering the spinal cord through the posterior nerve roots are very numerous, being about one and a third million in man; while the centrifugal neurons leaving the spinal cord through the anterior nerve roots number less than one-half a million.

The Centrifugal Neurons

The Lower Motor Neurons. From the nerve cells in the anterior horns of the spinal cord axons pass outwards constituting the anterior nerve roots and run, some to the muscles of the body, others to the ganglia of the sympathetic system and others to the glands.

The Upper Motor Neurons. From the nerve cells in the anterior central convolution (precentral gyrus) axons descend through the corona radiata and constitute the anterior two-thirds of the posterior limb of the internal capsule. Thence they descend through the crura cerebri and the pons and constitute the anterior pyramids of the medulla oblongata. Hence these upper motor neurons constitute what is called the "pyramidal tract," also called the "fasciculus cerebro-spinalis." From the lower part of the medulla a small part of the fibers of the pyramidal tract pass directly downward throughout the spinal cord, lying on the median surface of the anterior colunm (fasciculus cerebro-spinalis anterior or the direct pyramidal tract); while by far the larger part of these fibers of the pyramidal tract decussate and pass downward throughout the spinal cord in the lateral column of the opposite side (fasciculus cerebro-spinalis lateralis or the crossed pyramidal tract. The relative size of the direct and the crossed pyramidal tract varies somewhat and, in extremely rare cases, it has been claimed, no decussation of the pyramidal tract occurs. On the other hand in 15% of men decussation is complete and there is no direct pyramidal tract. The fibers of both the crossed and the direct pyramidal tracts (the fibers of the direct tract decussating in the anterior white commissure) finally ramify with the dendrons of the nerve cells in the anterior horn on the opposite side of the body from the cerebral hemisphere in which these fibers originate.

The cortico-rubral tract consists of fibers from the cortex of the frontal lobes to the red nucleus.

The cortico-pontile tract consists of fibers from the cerebral cortex to the pons.

The thalamo-spinal tract consists of fibers from the nerve cells in the optic thalamus, their axons passing downwards and terminating near the nerve cells in the anterior horns.

The rubro-spinal tract (Monakow's bundle or fasciculus intermediolateralis). From the nerve cells in the red nucleus axons descend, after decussation in the tegmentum, in the contralateral side of the pons, medulla and spinal cord, in which latter they lie in front of the pyramidal tract and hence this tract is some times called the "pre-pyramidal tract." They terminate near the nerve cells in the anterior horns.

The tecto-spinal tract. From nerve cells in the corpora quadrigemina axons descend, decussate beneath the aquaeductus Sylvii and pass downward through the formatio reticularis and the anterior and lateral columns of the spinal cord to the nerve cells in the anterior horns.

The vestibulo-spinal or Deiter's tract. From nerve cells in Deiter's vestibular nucleus axons pass downwards, mainly without decussating, through the anterior and lateral columns of the spinal cord to the nerve cells in the anterior horns.

The ponto-spinal tract. From cells of the formatio reticularis axons pass in part directly downwards in the lateral column of the same and of the opposite side of the cord to the nerve cells in the anterior horns.

The neurons of the last five of the above mentioned tracts constitute the extra-pyramidal motor tracts. They all terminate in the nerve cells of the anterior horns and have to do with automatic and associated movements, and muscle-tone. Lesions of these tracts are apt to cause motor disturbances, such as tremor, rigidity and athetosis, but no paralysis of voluntary motion, as long as the pyramidal tract is normal.

Between these bundles of centripetal and centrifugal fibers and connecting the former with the latter lie the central ganglionic masses of the spinal cord and brain, which receive the impulses coming through the centripetal fibers and shunt them along various different tracts of the centrifugal fibers, producing the relatively simple reactions of the spinal cord and the extremely complex reactions of the brain.

Although the cortical motor centers represent almost exclusively muscles lying on the opposite side of the body, it appears from clinical observation and physiological experiment that the muscles of the body have a bilateral cortical representation. By electrical stimulation of the cortex, the muscles on the same side of the body may be made to contract; although a much stronger irritation of the center is needed than is necessary to cause a contraction of the corresponding muscles of the opposite side of the body. Those muscles on both sides of the body which usually act together (diaphragm, etc.,) have especially well marked bilateral representation; so that these muscles are rarely completely and permanently paralysed in unilateral cerebral lesions. A cortical paralysis may abolish motion only and may be very circumscribed: two or three fingers, or the thumb alone. The actions which are especially lost in the cortical lesions are the purposeful actions which have been slowly acquired as the result of experience and training: actions which are peculiarly voluntary and skillful.

A destructive lesion of either the upper or the lower motor neurons will cause a motor paralysis. If the lower motor neurons are destroyed there will be a paralysis both of voluntary and of reflex acts: a flaccid paralysis with atrophied and degenerated muscles (252); while if the upper motor neurons are destroyed there will be a spastic paralysis of voluntary acts without muscle alterations; the reflex acts persisting and being increased: a spastic paralysis (251). (For explanation of the increase of reflex activity just mentioned, see page 11).

The Sympathetic System is not considered in this brief review of the physiology of the nervous system, because the Sympathetic System, important as it is, plays little part in the diagnosis of nervous diseases (Chart XVIId).

Movements (Charts IV, V, X)

When a sensory surface is irritated the animal often responds immediately by a comparatively simple movement, or the movement may occur only after a considerable space of time and may be very complicated, or it may never occur. Movements may also apparently occur spontaneously, not being preceded by any sensory irritation in the immediate past; although on careful analysis these spontaneous movements can always be referred back, indirectly, to some sensory irritation. All the actions of man or animal (for the day is past when the difference between man and animals was regarded as fundamental and not merely one of degree) have their origin immediately or remotely in sensory irritation or excitation, and all these different kinds of movements may be divided into two great classes: subcortical and cortical reflexes, according as the neurons concerned in the production of these acts have their cell bodies situated in the gray matter of the spinal cord and basal ganglia or in that of the cortex of the brain. Automatic acts are complicated reflex acts and may be either subcortical or cortical.

Subcortical Reflexes and Inhibition (Chart V)

The Simple Reflex. The centripetal nerve fibers terminate in the gray matter of the spinal cord and in that of the brain stem. Some of the nerve fibers, those conducting the impressions resulting from tactile, and especially those from painful, stimuli, terminate in synapsis with the dendrons of nerve cells lying in the posterior horns of the cord or in the sensory nuclei of the brain stem. Through these latter, the impulse is transmitted to a group of nerve cells in the anterior horns of the spinal cord or in the motor nuclei in the brain stem and along the axons of these latter cells to the muscles, causing them to contract and produce a motion which is called a reflex act. This group of nerve cells innervates not one, but a number, of muscles, in varying degree, to produce a definite purposeful movement and this resulting movement is to a degree orderly: "coordinated." The nervous complex just described may occur entirely through one segment, or metamere, of the spinal cord and is called the "unconditioned reflex," *i. e.,* not depending on other complicating factors or conditions. It is so simple that it occurs very rarely, perhaps never, in man; although it does occur in all its simplicity in some of the

lower animals. It occurs in frogs and most clearly in those whose spinal cord has been separated from the brain by a transverse cut at any point above the reflex arc involved in the act. A reflex act is a reaction from an irritation, which under like conditions always take place in exactly the same way; it seems purely mechanical, as if a machine were working. The irritation may be a usual (normal or adequate), or unusual (abnormal or inadequate) one; the former being much more effective; and it may arise from stimulation of the skin, mucous membrane, muscle tendon or fascia, or of any of the organs of special sense. The centripetal neuron, the centrifugal neuron and the connecting neuron joining together the two others form what is called the spinal or, including the brain stem, the subcortical reflex arc. It is the simplest and most primitive form of nervous reaction and is the type, or pattern, of all other more complex forms of nervous reaction. For its production there is necessary a *receptor* (end-organ), a *conductor* (neurons) *and an effector* (muscle or gland). By the neurons the receptor and the effector are brought into intimate connection. Usually, if not always, many groups of nerve cells lying in different levels and the coordinating influence of the cerebellum take part in the ordinary reflex activity. Such simple reflex acts are the only ones occurring in the body during the early months of life and are, at first, unconscious acts, and, indeed, many reflex acts occur unconsciously throughout life (pupillary, etc.). Similar reflex acts cause the respiratory and cardiac movements, the flow of saliva and other secretions, the vascularity of organs and consequently the warmth of the body, and in general regulate the physiological actions of the body. These are called *instinctive* or hereditary actions; the result of the experience of ancestors and, therefore, called *phylogenetic* in contradistinction to *ontogenetic* acts, which are acquired by the education and experiences of the individual. Muscular tonicity is a variety of reflex action and disappears in destructive lesions of any part of the reflex arc (240).

The groups of nerve cells lying in the anterior horns and causing, when in activity, a definite coordinated movement are called "common paths." These are the paths by which nervous impulses coming from different parts of the body leave the spinal cord to produce the reflex act and are the paths which all these nervous impulses have in common; while the sensory impulse arising from irritation of any one sensory surface is called a "private path," and is at the service of only one group of end-organs.

The reflex act is influenced by many other conditions. Slight irritative lesions of the reflex arc cause exaggeration, while destructive lesions cause abolition, of reflex action. If the neurons in the cord are excited by a strong painful irritation of a peripheral nerve, as for instance the bathing of a sciatic nerve of the frog in a strong salt solution, the reflex acts will not take place. It is said to be *inhibited*. It can also be inhibited by strong impulses coming down to the cord from the higher nerve centers. Finally, a reflex act during its occurrence inhibits, more or less completely, all other reflex activity of the spinal cord, and especially inhibits the activity of the antagonistic muscles.

The *voluntary* abolition of reflex activity (inhibition) may be brought about by a contraction of those muscles which antagonize the muscles taking part in the reflex act, or this latter act may be "inhibited" by a direct action upon the subcortical motor cells restraining them from taking part in it, nullifying their activity. In addition to this voluntary inhibition, a great variety of nervous activities taking place in almost any part of the nervous system (especially strong, painful impressions), and even the normal process of cerebral activity will cause a more or less complete inhibition of reflex activity of the lower parts of the cord. *Complete* destruction or section of the upper part of the spinal cord is said to abolish all reflexes in the lower part. This phenomenon, if it really exists, except as a temporary one due to shock, etc., has not been satisfactorily explained and is in marked contrast to the exaggerated reflexes found in incompletely destructive lesions of the upper portion of the cord, and appears from the most recent observations to have no foundation in fact.

Conduction of reflex or other impulses along the peripheral nerves is equally rapid whatever may be the intensity, or quality, of the irritation, but conduction through the gray matter is much slower and varies greatly with the intensity and quality of the irritation. The gray matter also possesses the power of summation; so that excitations too feeble to give rise to a reflex may be-

come potent ones by repetition at very short intervals of time. The gray matter immediately following its activity shows a "refractory period" of longer or shorter time, during which it is inexcitable or exhausted. This indicates that the gray matter accumulates energy during rest, which it discharges when in activity. This refractory period may play its part in rhythmical action; such as the heart-beat. Most reflex acts are purposeful and healthful in their nature. Many of them are absolutely essential for life. They may be divided into the offensive and defensive.

A destructive lesion of any portion of the reflex arc causes abolition of the reflex acts, as does also a strong irritation of the higher nerve centers. Slight irritative lesions, such as slight inflammations, involving any portion of the reflex arc, will cause an exaggeration of the reflex act, as will also, and more commonly, a lesion which interferes with conduction of nervous impulses (inhibitory impulses) through the central motor (cortico-spinal) neurons. An irritation, especially a continuous one, even if not very intense, will often cause a tonic spasm or contracture.

Coordination (43, 248, 638)

The centripetal neurons, which enter the cord through the posterior horns, in part, as we have just described, run forward and passing through a connecting neuron to a motor neuron in the anterior horn form a "reflex arc." Other centripetal neurons run upwards in the spinal cord in various tracts, already described on previous pages, to the higher nervous ganglia lying within the skull. Some of these ascending neurons pass, directly or indirectly, to the cerebellum, which is the great coordinating center of the nervous system. As we have learned, the simple reflex act is coordinated, a number of muscles being concerned in the act, some contracting powerfully and others with varying degrees of intensity. This is due to the fact that the nerve cells in the anterior horns are arranged in groups, the starting point of the "common path;" the receptive neuron being a "private path." Each of these common paths, when incited to action, produces a definite coordinated movement. This coordination is very simple and applies only to one action. Most movements of the body consist not of one but of a series of coordinated movements in definite sequence. This complicated coordination, both for reflex and for so-called voluntary acts, takes place in the cerebellum and in lesions of the cerebellum both these kinds of actions become incoordinate, irregular and ineffective. The subcortical reflex is congenitally coordinated (phylogenetic). On the other hand, coordination due to cerebellar influence is acquired by experience, training, practice (ontogenetic). The cerebellum coordinates a series of reflex acts in definite coordinated succession.

The mechanism of the action of the cerebellum is very little understood. As already described, the centripetal fibers from Flechsig's tract (the direct cerebellar tract) and from the nuclei of the columns of Burdach and Goll pass through the restiform bodies to the Purkinje cells in the cerebellar cortex. From the semi-circular canals, through the vestibular nerve, fibers run to Deiter's nucleus. Centrifugal fibers run from the dentate nucleus of the cerebellum to the red nucleus and from the nucleus and from Deiter's nucleus fibers descend to and through the spinal cord in the extrapyramidal tracts already mentioned. The cortex of the cerebellum is connected with the cerebral cortex by centripetal fibers through the middle peduncles. Centripetal fibers also run from the dentate nucleus of the cerebellum to the red nucleus and thence to the cerebral cortex of the frontal lobes (the cerebello-rubro-frontal tract). The cerebellum has, therefore, very wide connections. From recent investigations there seem to be definitely localized coordination centers in the cerebellar cortex similar to those long since demonstrated to exist in the cerebral cortex.

The Cortical Reflexes

In addition to the ascending neurons passing to the cerebellum, others ascend to their final termination in that portion of the cerebral cortex which lies posterior to the fissure of Rolando, being interrupted in their course by various ganglia at the base of the brain. These so-called "sensory projection fibers" which spring from definite sensory end-organs terminate in definite and distinct cortical areas. Thus, the fibers from the retina (optic fibers) terminate in the occipital lobe; those from the nose (olfactory fibers) and those from the mouth (gustatory fibers) terminate in the cornu Ammonis; those from the ear (auditory fibers) terminate in the anterior trans-

verse temporal convolution, those from the skin (tactile fibers) terminate in the posterior central convolution and those from the muscles (muscle sense) terminate in the inferior parietal lobule. These localized areas constitute the anatomical basis of cerebral localization (Figs. 15 and 16). They are all situated in the hemisphere contralateral to the peripheral sensory organs from which their long projection nerve fibers spring (having passed through sub-cortical ganglia in their course). These areas are all connected together by nerve fibers (axons) which connect every portion of the sensory cortex with every other portion. These are called "association fibers." Finally, from each of these localized differentiated sensory areas, bundles of axons pass forwards under the fissure of Rolando to the anterior central convolution and its immediate neighborhood (the so-called motor area of the cortex) and come in contact with groups of neurons, the axons of which constitute the pyramidal tract and terminate in those groups of nerve cells in the anterior horns of the cord which we have already described and to which we have given the name of the *common path* along which impulses pass to the various groups of muscles. These groups of neurons whose cell bodies lie in the anterior central convolution (the motor area) may be called the common path for the impulses coming from the sensory area of the cerebral cortex. It is quite possible, then, for a nervous impulse entering the spinal cord through a posterior nerve root, not only to pass forward to the motor neurons in the anterior horns and produce a reflex act, but also to ascend by the sensory projection fibers to the cerebral cortex posterior to the fissure of Rolando and there to be reflected to a common path in the anterior central convolution and thence downwards along the pyramidal tract to the same common path in the anterior horn through which the spinal reflex impulse passes.

The gray matter of the cord is compressed into the center of the cord, of which it forms a long continuous "H" shaped axis. Its transverse area is so small that an impulse entering through the posterior roots finds a short and easy route to the common path and passes along it so quickly that it is not apt to be modified or inhibited by any other impulse reaching the common path at the same instant. Subcortical reflexes are, therefore, quickly and definitely performed and are relatively simple in character.

The gray matter of the cerebral hemispheres, on the other hand, is not compressed into their centers, but is spread out in a large area over their entire surfaces, which are themselves, greatly increased in extent by their numerous sulci. When an impulse reaches the cerebral sensory cortex through the sensory projection fibers, it may pass, as we have said, to a common path in the motor cortex. It may also pass through the association fibers to many, widely separated, areas of the sensory cortex and set their neurons in activity. Through these latter numerous neurons, impulses may also pass to the same common path, some tending to increase, others to impede or abolish, its activity. The cortical reflexes are, consequently, slower than the subcortical and do not follow immediately upon the excitation. They may, indeed, be delayed days, weeks, even years after the time of the original excitation which was the primary cause of them. They may never occur. They may be exceedingly characteristic individual acts very different from the stereotyped reflex acts and may consist of a great variety of successive acts.

Just as the subcortical reflexes, so the cortical reflexes have been the object of much experimental work. One of the earliest of these recent series of experiments was performed by Pavlow. His results, stated briefly and without details, are that the salivary secretion of a dog, which is normally caused by food in his mouth, can be excited, after a course of training or education, by optic excitations (one of various colors or of different intensity of the same color), or by auditory excitations (one of various sounds or different intensities of the same sound), without food in any way entering into this final acquired reaction. This reaction will not occur if the corresponding sensory cortical area be destroyed. A very slight sensory excitation occurring at the same time will *abolish* or impair it. It is a cortical reflex but has in large degree the characteristics of the spinal reflex. It is called the "conditioned reflex" in contradistinction to the "unconditioned" reflex: the normal response to food. The conditioned reflex is not acquired by punishment and reward, but merely by association and, when acquired, does not result in pleasure or pain. It is a true reflex with no emotional content.

Somewhat analogous reflexes (sometimes called motivated reflexes) have been acquired by

animals as the result of long training by means of punishments and rewards; such as rats finding their way through a maze and the training of animals in various ways, and more broadly in "forcing the formation of sensory habits." Much and most fruitful work has been done in this line. The human being undergoes a very similar training, directed by rewards and punishments, in the home, the school and in life.

The cortical reflexes, often called the "association reflexes," and known by a still older designation, "voluntary movements," are the result of education. Given the knowledge of a person's education, and enviroment in all its details, his acts can be predicted with a great deal of certainty and are practically so predicted by his fellow men, even though they do not know all the details of his heredity, which knowledge is, of course, also necessary for an infallible judgment. (See voluntary movements.)

The subcortical reflexes, the simple reflex acts, may be entirely unconscious ones. They are most marked when by a lesion the spinal cord in its entirety or its lower portion is separated from the brain, and in such cases (which are of not infrequent occurrence) the individual has no consciousness of the movements of his arms, or legs or sphincters, unless he is looking at them.

The cortical reflexes, on the other hand, are usually associated with very remarkable phenomena (including consciousness), which we have next to discuss.

Before doing so it may be noted, as a summary of previous statements, that there are three ways, and only three, in which an animal responds to the various stimuli coming from the external world:

1st. *Reflex Acts* which are centered in the spinal cord and brain stem, and which are relatively slightly, or not at all, modified by other stimuli.

2nd. *Automatic Acts,* centered in the corpus striatum and nucleus lenticularis and greatly modified by other stimuli including the emotions.

3rd. *Volutary Acts,* centered in the cerebral cortex and profoundly modified by many stimuli arising from present and past perceptions, matured judgments, habits and emotions.

The "So-called" Psychic Functions (Charts III and XVI)

So far in our discussion of cerebral activity (the excitation of the sensory cortex and the conditioned, motivated and associated reflexes which result), we have been dealing with facts which can be definitely proved by anatomical and physiological investigation. What we have considered however, does not, by any means, include all the phenomena connected with cerebral activity.

When a nervous impulse reaches the sensory cortical area it produces, if of sufficient intensity, a phenomenon called sensation. Thus, when we are looking at a tree we appreciate something which we call the sensation of the sight of a tree. It is often called the image of a tree although there is in the brain nothing like an image on a photographic plate of the tree, but rather a cortical activity which is the symbol of a tree. Moreover, while we are looking at the tree, there is taking place another cerebral activity of which we are entirely ignorant and unconscious—a permanent change occurs, whether static or dynamic, which constitutes a memory of the tree; so that when we close our eyes an image of the tree may remain, which we clearly see and which is perfectly true; although it does not possess the quality of reality. This memory of this identical tree remains permanently in the brain, potentially, and can at any time be brought into consciousness (a term later to be defined) by a process which is called the association of ideas (also later to be defined).

Sensation and memory are the basic factors in psychology and upon them is built up the elaborate structure of this science, whether it be called introspective or physiological psychology. Sensation undoubtedly is a manifestation of force, the product of the oxidation of the nutriment in the blood and ceases as soon as the blood ceases to flow in the corresponding part of the cerebral cortex, but no psychologist or physiologist has ever satisfactorily, or even intelligibly, defined what sensation really is. This is true of many of the other natural forces. To mention only two: gravitation and electricity have never been explained. Their essential nature, after centuries of observations, is not yet understood. This does not prevent our studying them and discovering

that they invariably act in certain well defined ways, that they follow certain laws; and by availing ourselves of our knowledge of these laws these forces have become our very useful slaves. It may be that in the future we shall discover the essential nature of these forces more or less completely, but that day is not yet. We may also discover what sensation is, but it can even now be studied, and as a matter of fact this form of energy is being experimented with in every psychological and physiological laboratory in the land.

The Psychology handed down to us by our fathers attempts to solve these facts by traditional authority, by abstract reason and by introspection and has introduced the element of the supernatural and of mystery. The result after thousands of years of this study has been far from satisfactory. Our knowledge acquired by these means has advanced little, if at all, beyond that of the ancient Grecian philosophers. To the physiological psychologist of recent years these facts still await solution; but to him they are the result of cortical activity and do not require, and probably will never require, a supernatural explanation. What sensation is, has not been explained either by the scientist or by the philosopher. To the former, however, it appears to be energy produced by cortical activity.

The mystery of the mechanism of the animal body is being slowly dispelled by investigation. This investigation has been carried on, and is being carried on, under many difficulties. The dissection of the human body and experimentation on animals is still repugnant to many. The prejudices of mankind have to be reckoned with. Many highly educated men, while granting that most of the organs of the human body are in their activity subject to ordinary physical laws and that the source of their activity is the nutriment in their blood supply, are, yet, unwilling to grant that the activity of the nervous system is to be explained in the same way. They point out that most of the internal organs take certain chemical substances from the blood and convert them only into other chemical substances and that the chemist in his test tubes and other apparatus can imitate these reactions more or less perfectly. They omit to state, however, that there is one set of organs, the muscular system, much more extensive than any other organs in the body, which takes the nutriment from the blood and transmutes it into a form of energy, contractility, and, thus, does exactly what the nervous system does, when it transmutes the nutriment in the blood into forms of energy called the sensation, consciousness, etc. The reason that I am devoting what may appear to be too much space to this subject is that I am desirous of presenting the physiology of the nervous system on a physiological rather than on a mystical basis; and, yet, what is here presented is but a bare outline of the subject and is in need of much amplification.

Sensation and consciousness, whatever their nature may be. are the fundamental elements in any aspect of psychology or of cerebral physiology, if, indeed, any distinction can be drawn between these two sciences. They are the "Axiom" or the "Given" of mathematics, of logic and of psychology. They are self-evident propositions which cannot be further analysed.

Sensation (Charts VI and XIV)

When the various impulses originating in the sensory end-organs have passed along the various tracts and have traversed, and been interrupted by, several masses of gray matter, they reach the sensory area of the cerebral cortex and there give rise to a new form of energy called sensation. That is to say, a physical force (as for instance, ether in rapid undulation) is converted in a terminal organ into nervous energy, and as such, having traversed the sensory tracts, reaches the cerebral cortex. It is there transmuted into a new form of energy (as for instance the sensation of light). The sensation of light takes place in the brain, not in the eye, and has no similarity to the undulations of ether from which it normally originates, and it may, indeed, be caused not only by these, but also may originate, in perfect darkness, from mechanical irritation of the eye (as by pressure from the finger upon the eyeball) or of the optic nerve. Sensation is, therefore, rather a symbol than a picture (the image of the older psychology) of the external object, with which by experience it is associated.

Sensation is thus a special, individual form of energy, similar to electricity, light, etc., which is produced in the cerebral cortex and which has its special individual characteristics. It has an

analogy to electricity produced on amber by friction. Sensations originating from the different organs of sense are located, as we have seen, in different and special portions of the cortex (Figs. 15 and 16) and do not at all resemble the external phenomena causing them. A clap of thunder and a flash of lightning are very different external phenomena, but the reactions in the cortex, which constitute these sensations, probably vary in topography rather than in quality. We know nothing more of the essence of this form of energy, which we call sensation, than we do of the essential nature of electricity, or of contractility in the muscle fiber or in the amoeba. We know something of its effects and we know something of the locality of the cerebral cortex in which it occurs (Figs. 15 and 16) and that it is very dependent upon abundant blood supply and we believe that it depends upon physico-chemical actions taking place in the cortex. Conscious sensation, probably, occurs only in those animals which possess cerebral hemispheres.

Sensation and all other forms of mental activity are absolutely dependent upon a fairly healthy cerebral cortex and a fairly abundant blood supply to it. When the cerebral hemispheres in an animal are removed, or when the cerebral cortex in man is entirely, or mainly, destroyed by disease, or in a child the hemispheres are absent or very defective, or when the blood supply is cut off from the cerebral cortex altogether or in large part, then sensation, perception, memory, thought, emotion (and its corporeal expression, except in anger), ethics, association of ideas, voluntary motion, inhibition, intelligence, personality and consciousness are all lost.

Sensation is the simplest manifestation of consciousness (see later) or cognition. For its production a certain degree of intensity of the nervous impulses is essential (the threshold), below this point of intensity the cortex may be in activity, but sensation will not result; the activity will be subconscious. A series of slight subliminal impulses, quickly repeated, may by summation cause sensation. There is, therefore, a minimum of intensity necessary for sensation; just as electricity passing through a wire must have a certain intensity before the wire glows and light is produced. There is also a maximum beyond which, no matter how great the irritation, there is no increase of sensation, but rather a diminution from exhaustion of the nerve cells. Between this minimum and maximum point, sensibility increases, or diminishes, not continuously, but by little steps: a definite ratio to the stimulus, in accordance with Weber's law, which, although not absolutely, is approximately, correct, especially for stimuli of moderate intensity.

Furthermore, a weak or moderately strong excitation may reach the cortex at a time when other portions of the cortex are in such strong excitation that this weak irritation may produce no sensation, but remains subconscious, is inhibited by the stronger cortical activity. The line between the conscious and the subconscious cannot be sharply drawn. Subconscious cerebral activity is much greater in volume, although much less in intensity, than is cerebral activity resulting in consciousness. *Sensation is the inter-reaction between a relatively strong nervous impulse from an organ of sense and the cortical activities constituting the memories of those previous perceptions, emotions, etc., which are the individual's personality.*

Perceptions and Concepts (Chart VI).

A perception consists of a combination of sensations, which are obtained from various sensory end-organs, all of which proceed, usually simultaneously, from the same external object. A perception of an apple is composed of several sensory impulses: of visual sensations caused by nervous impulses from the retina, representing the outline and markings and color of the apple; of muscle sensation caused by nervous impulses from the ocular muscles, representing its distance from the eye, its position in relation to other objects and to some extent its form; of tactile sensations caused by nervous impulses from the hand, representing its form, firmness and texture; and of gustatory sensations caused by nervous impulses from the mouth, representing its taste. The various physico-chemical changes, thus set in activity in the cortex, combine to produce the full perception of the apple, which perception is greatly modified by our emotions, moods, memories and the then present other perceptions, and is not exactly the same, sometimes widely different, in different men. The perception of an apple by Adam was probably very different after the fall of man than before it. For a full and complete perception, consisting as it does of so many elementary sensations, quite an appreciable time, or frequent repetition, is needed. It is quite pos-

sible that a full perception may consist both of conscious and subconscious elements. The kinesthetic sense, for instance, is rarely, if ever a conscious sensation. The development of a perception is found by experiment to proceed from generalities to details. A combination of the full perceptions of many apples, each resembling and at the same time in some respects differing from the others, produces the idea or *concept* of an apple, with which is associated its written or spoken name and any other experiences or knowledge which have become associated during our life with apples. (See also under Association.) A concept is, therefore, an abstraction.

Perceptions occur in the cerebral cortex in immediate proximity to the cortical termination of the corresponding projection fibers. Each cortical center consists of a smaller portion, in which the projection fibers terminate, and a larger portion, in which perceptions take place and in which their memories are stored. Thus, the optic fibers terminate in the lips of the calcarine fissure, while the rest of the median and convex surface of the occipital lobe is devoted to optic perceptions and memories (Figs. 15 and 16). When sensations only, but not perceptions, can occur, as when that portion of the center in which the projection fibers do not terminate is diseased, the condition is called, in general, *agnosia.* When there is a failure of tactile perceptions the condition is called *astereognosis;* in failure of optic perceptions, *soul-blindness,* or *psychic blindness,* and in failure of auditory perceptions, *soul-deafness,* or *psychic deafness,* or *auditory* or *sensory aphasia.* When that portion of any sensory area of the cortex in which the sensory fibers terminate is diseased, both perception and sensation are abolished.

Memory (Chart III)

When perception takes place, physico-chemical changes are occurring in a definite portion of the cerebral cortex, which not only produce the perception, but also leave, thereafter, a permanent alteration in the cortex (a *vestige*). The energy derived from the chemical changes taking place in the cortex during an active perception may result in a structural, physical or chemical change in the nervous elements, or more likely in the storing in them of potential energy, which can be liberated and become actual later. Memories seem to be dynamic changes in nerve cells and fibers which reduce the resistance to subsequent similar impressions or excitations. Certainly, a definite change is brought about which registers a permanent memory of the object perceived and subsequently this memory can be latent or active from time to time. Consciousness, the actual perception of an object and its associated active memories (active attention), is a very exhausting, energy consuming process for the cerebral cortex. Subconsciousness, and the preservation of memories, not actually present in consciousness, are not exhausting to the cortex, even though the memories be preserved for many years.

In virtue of this change in the cortex, a memory of the corresponding object always results from excitation of this altered cortex. The memory may be aroused, or enter into cognition, by the external force which originally caused it and, then, the object will be recognized (re-known), because the actual perception corresponds perfectly with its memory; or the memory may be aroused by way of association fibers which the original perception had previously set into activity. Memories become associated with each other in accordance with the relationship of the objects causing them, as the result of our experiences with these objects. In perception, then, a trace of the cortical excitation remains in the cerebral cortex as a memory, in a sense analogous to the persistence of the after-image in the retina after strong excitation (looking for some time at a bright light). The retention of any memory depends upon the frequency of the perception causing it, its interest and its startling nature. The recall of a memory is by way of the association fibers from a perception or some other memory associated with it.

In comparing an actual perception of an object, or an experience, with its memory, if the two exactly coincide and resemble each other, there is not only the act of recognition, but also what is called "identification" and "similarity;" whereas if the perception and the memory of a former perception do not exactly coincide, there is what is called "non-identity" and "dissimilarity," and this action is called "discrimination." This cerebral activity with its two results: identification or discrimination, is the basis of classification, systematizing and judgment.

Memories are, however, very different qualitatively from the original perceptions. The former have no actuality. Even though they may be at times very vivid, they never seem real to a normal personality. This is due to the absence of any nervous impulses coming simultaneously from the peripheral sense organ, especially from its musculature, which were present in the original perception. They are never so intense as the original perception. It is almost, if not quite impossible to recall a memory derived from any peripheral organ of sense, while that organ is engaged in producing an actual perception; the actual perception is much too strong for the memory and the latter is inhibited by the former.

The cortex of the brain is in great part a huge store-house of memories. The memories of any object vary in different men according to their training, education, attention and former associations with the object. These memories are grouped together; so that those which are derived from the same organ of sense lie together in the cortex. This localization of memories has been worked out with great care and is to a considerable extent known. It is shown in figures 15 and 16. A local cortical lesion may thus produce a loss of a group of allied memories.

All memories are sensory in character with the exception of one extremely doubtful group. This group consists of memories of so-called "innervation feelings." When a muscle is contracted the person to whom it belongs has a feeling of this contraction and can estimate its strength. Such innervation feelings, if existent, are sensory, but it is difficult to be conscious of them. They are, in the main, subconscious. This feeling is called an "innervation feeling" and its memory, it is claimed by some investigators, is stored away in the cortex of the anterior central convolution and especially in the neighborhood in front of it. This innervation feeling, it is claimed, is essential for the voluntary performance of the corresponding act. When, in consequence of a cortical brain lesion in the area in which these memories are stored, a person loses the power of performing certain acts, he often says, "I have forgotten how to do it." These innervation feelings and memories, if they really exist, do not obtrude themselves strongly into our consciousness. They have rather to be sought for. It is, indeed, very difficult if not impossible to be conscious of them. They may, however, be subconscious and play their part in the production of complex perceptions.

There are many physiologists and psychologists, however, who question whether there are any so-called innervation felings or memories, and regard the cortical motor cells as merely "common paths" similar to those in the spinal cord. However that may be, whether true innervation memories exist or not, the function of this cortical area is an actuality and whenever a portion of the motor cortex is sufficiently excited by a cellular activity, a perception, or a memory, in the sensory cortex so-called voluntary, or association, action results. (See Voluntary Movements).

Consciousness and Subconsciousness (Charts III and IV)

Consciousness and subconsciousness are terms used to express the fact that the cerebral cortex is in activity. Although convenient expressions, they should not be regarded as representing distinct, possibly conflicting, entities. They are both component parts of the personality. The difference between them is only one of degree: the degree of intensity of the local cortical activity which produces them combined, perhaps, with a sense of "friction" which the intense activity meets in passing through the cortical neurons. As this resistance decreases with repeated use an act, at first conscious, may become subconscious. A person is at times unconscious of his habits: his habitual acts. At one instant a sensation, a perception, a concept, an idea, a thought, a judgment or some other product of cerebral activity is present in consciousness because of a strong local cortical activity and the next instant the activity becomes less, is replaced by another strong local cortical activity, and the former ceases to be present in consciousness and becomes a part of the subconscious cerebral activity. There is, thus, a constant interchange between local cortical activities and consequently the content of consciousness is constantly changing. The fact that an intense cortical activity makes its resulting energy an integral part of consciousness, makes it also a dominant factor in any judgment or action which may result. Normally, during life, there is constant cortical activity, whether it is an activity resulting in consciousness or subconsciousness. Furthermore, there are all degrees of intensity of this activity and consequently varying de-

grees of consciousness from vivid expectant attention (concentration), to passive attention (reverie) to semiconsciousness, to stupor, to dreams (by night or by day), to subconsciousness and, in the absence of all cortical activity, to unconsciousness. What consciousness really is we know no more than we know what it is which is exhibited by amber when it is electrified by friction. Yet we do not hesitate to say that electricity is a form of energy, or force, produced by friction upon amber, by a dynamo, or by numerous other means; and just as we say that the potential energy of coal may be converted into electricity; so we may say that the potential energy in the blood (derived from food) is converted in the cerebral cortex into conscious sensations, perceptions, etc., or, taken collectively, into consciousness.

Consciousness consists, at any instant of time, of the then present perceptions and of those past memories which are directly or indirectly associated with these present perceptions and which have been awakened by them into activity at that instant of time. Consciousness is thus a form of energy resulting from a high grade of activity of the cerebral cortex. The other memories, at that instant active, but not active with sufficient intensity to enter into consciousness, constitute subconsciousness and may at any time become conscious memories. The content of consciousness embraces only a small fraction of those activities which take place in the brain and indeed only a fraction of those activities which take place in the cerebral cortex at any instant of time.

The cortical activities constituting consciousness are very much less numerous and extensive than those constituting subconsciousness. When we first dress each morning, we are conscious of the contact of our clothes with our skin, but soon this sensation ceases, is replaced by others and the cortical activity caused by the contact of the clothes becomes subconsciousness. It still persists, however, and if by way of association fibers its activity be slightly increased it will again enter consciousness and the sensation will again arise temporarily. We walk in the street and are conscious of seeing and hearing many things, but many other things cause cortical activity of such low intensity that we fail to be conscious of them. They remain subconscious and a faint memory of them persists which may later be brought into consciousness by way of the association fibers, the impulses from which may increase the subconscious activity to a degree which constitutes consciousness. A familiar example is that of a man whose attention is so fully occupied that he does not hear the striking of a clock, but later recalls the memory of this sound, which memory was obtained subconsciously.

Consciousness and personality can probably be best studied in their forming in infancy, and in such a study they may well be found to consist essentially in the action and interaction of each new perception on the few other perceptions previously acquired, which at that date constitute the rudimentary personality of the child.

A new born babe has, probably, no consciousness. An infant attains consciousness slowly as he gradually obtains perceptions and memories and forms a large number of associations of all kinds. When an infant has his first perception this one percption, together with certain rudimentary sensations he may have acquired, constitutes his entire consciousness and his entire intelligence. It is all he knows. As other perceptions are obtained and associated together his intelligence and his consciousness become larger, more distinct and more complete. The child in his development increases with great rapidity the number of his perceptions, less rapidly and subject to many subsequent corrections his associations, still less rapidly his concepts, even more slowly his ethical and aesthetical ideas, and yet more slowly his abstractions. But at the end of a few years any perception or memory suggesting action is subjected to the interplay of all these activities before the action is done or left undone. In other words, consciousness and the act of thinking and reasoning on which action is based (see Voluntary Motion) are already, even in childhood, very complicated, perhaps more so than in later life, when action is mainly determined by habits of thought and by judgments firmly established by numerous experiences.

Consciousness is a form of energy, such as light and electricity, which is transmuted from other forms of energy by physico-chemical action taking place in the cerebral cortex. Consciousness embraces all those chemical activities which have a certain intensity; below this degree of intensity the activities constitute subconsciousness. The process is somewhat analogous to that in an

electric light bulb through which an electric current may be constantly flowing, but which only gives forth light when the current has attained a centain intensity.

Consciousness is constantly being newly formed and is dependent upon the perceptions, memories, feelings and ideas, ethical and others, present at any one instant. These phenomena themselves constitute and are consciousness. None of them, usually, continues. Others are constantly replacing them. None remains constant. The continuity of consciousness is preserved by the mingling of memories of past perceptions with present ones, and by memories of past states of consciousness.

Consciousness has narrow limits. It cannot contain many perceptions, ideas, etc., at the same time. We daily take advantage of this. When a child or an adult is conscious of a painful perception or idea and is consequently unhappy we suggest to him another perception or idea and emphasize it until it replaces the painful one, which sinks into subconsciousness and is less dominant.

Just as reflex action taking place in the spinal cord temporarily inhibits more or less completely the activity of the rest of the cord; so a strong activity in the cerebral cortex tends to inhibit the activity of the rest of the cortex and to dominate consciousness. This strong local cortical activity is accompanied by a local increased blood supply, as can be shown by the thermopile. The blood supply is, therefore, in constant, more or less rapid (usually rapid) ebb and flow throughout the different areas of the cortex, being most abundant in those areas which are in activity.

Consciousness is a condition which, as yet certainly, we do not understand, although in a general way we regard it as the result of chemical changes taking place within the cerebral cortex. The chemical changes themselves are not consciousness, but they produce this form of nervous energy very much as a steam boiler and a dynamo, or a galvanic battery, produces electricity. We are as ignorant of the exact nature of consciousness as we are of that of electricity. These chemical changes produce consciousness (or rather the perceptions and emotions which constitute it), a form of nervous energy; just as the chemical changes taking place in any living cell or tissue of the body produce a form of energy peculiar to itself. Consciousness is thus locally and continually produced in the brain. The cortical activity causing it passes according to definite channels to other regions of the cortex, surges through the brain, awakening memories and ideas and causing actions and reactions. The subject is a most difficult one and is made, in a sense, more difficult by the ambiguities of language, which allow us to replace an idea by a word of somewhat uncertain definition and thus leads to uncertain and faulty reasoning, or to a high sounding sentence which means nothing. Consciousness, or cognition, seems to be something added on to the essential processes taking place in the brain. The various association reflexes occurring in the brain could take place and do take place quite accurately without consciousness, as for instance in the automatic acts of the somnambulist, or in the epileptic trance.

That consciousness has any power to influence cerebral activity and the association reflexes is not evident and has not been proved. That frequently it cannot do so is proved by the oft recurring condition where an idea is present in consciousness which the person is anxious to dismiss but his consciousness even combined with his so-called will power is unable to dismiss it. Only when by the association fibers a different and stronger local cortical activity is awakened will the distressing idea fade away.

The brain is very abundantly supplied with blood, especially the cortex, and the latter is very sensitive to any interference with its blood supply. Loss of consciousness, which occurs normally in sleep and pathologically in many conditions, is caused much more frequently by a change in the quantity(anemia) or quality(drugs and poisons, including sepsis and other autogenetic toxic products) of the blood supply than by all other conditions combined. Perversions of consciousness, on the other hand, seem to depend less upon the quantity of the blood supply than upon its quality (poisons) and upon changes, organic or functional, in the cerebral cortex, especially upon its exhaustion.

Finally it is to be remembered that the cortical cellular activities not only accompany (are the cause of) but precede sensation. consciousness and the other "psychic" functions. The con-

ditioned reflexes are cortical activities, but they occur in infants and animals unconsciously and prior to any evidence that the "psychic" functions as yet exist.

Emotions (Chart III and IV)

The great majority of sensations perceived by an individual during his life are followed by the reactions in the nervous system already described. There are, however, sensations and perceptions which are accompanied by an emotional content—pleasure or pain, according as their content suggests the well-being of the body, a richer and happier life on one hand; or death, or a restricted and unhappy life on the other hand. Heredity, early education, past experiences and associations, also, influence the emotional content of a perception. Such perceptions with an emotional content, cause an abnormally powerful reaction on the part of the nervous system. The emotional reflexes are among the earliest to be observed in the infant. It appears from experiments on animals that the emotions of pleasure are the result of,or at least are influenced by,cortical activity; while the emotions of pain, anger and displeasure occur after the removal of the cerebral hemispheres and are the result of the activity of the basal ganglia. These emotions produce a striking effect upon the functions of the abdominal viscera as has been abundantly proved by animal experimentation and human experience. Thus, pleasant sensations promote the activity of the muscles and glands of the stomach: while unpleasant sensations retard, or entirely arrest, this activity. But, by far, the most striking and important influence of the emotions upon the somatic activities is that manifested by anger and fear upon the secretion of the suprarenal glands and the consequent rapid and excessive production of adrenalin. The emotions of anger and fear are usually followed by violent muscular activity whether in the form of fighting or flight. It has been proved by experimental and clinical evidence that adrenalin appears in increased quantity in the blood within a few seconds or a few minutes after the occurrence of violent anger and fear. Furthermore, it has been proved by experiment that adrenalin in excess in the blood causes *first* an increase in the amount of sugar in the blood which is essential for muscular contractions; *second,* a dilatation of the bronchioles and increased rapidity and power of the heart action, thus causing an increased amount of oxygen in the blood which is also essential for muscular contraction; *third,* prevents muscular fatigue and restores power to exhausted muscles, and *fourth,* quickens coagulation of the blood. All of these conditions are of great practical value to the angry or frightened animal, whether in a fight or in a flight.

In addition to these violent emotions, there are more gentle emotions of pleasure and pain which we call "feelings" and which are due in part to internal, or general, sensations from the body itself and in part from the success or failure of our undertakings in life. When the bodily functions are disordered we have a general feeling of discomfort and when all is working well we have a sense of buoyancy and exaltation; all moves smoothly without friction, as in a well oiled machine. These internal sensations, as was mentioned on a previous page, are ordinarily the dominant factor in our feelings and emotions and greatly influence also our voluntary actions, which for instance may be altogether different in a state of hunger from those in a state of satiety. Indeed the internal sensations, such as hunger, etc., are very often themselves the cause of extensive voluntary acts, which usually result in the relief from this sensation. As these internal sensations vary from time to time, our moods change, and perceptions, which at one time are pleasant, may at another time be unpleasant. Irrespective of our moods, however, some perceptions are almost always pleasant, others are not. Things which tend towards the preservation and health of oneself and one's family are usually pleasant and vice versa. Perceptions to which we have become accustomed are usually pleasant, and even unpleasant perceptions by frequent repetition at times become bearable and even pleasant.

Not a few perceptions are accompanied with relief or discomfort, as when hunger is assuaged, or we accomplish something desired, or in some way contribute to our well-being or success and thus give pleasure; while other perceptions act in a contrary manner. These feelings of pleasure and pain may be due in part to the intensity of the sensation or perception; in part to

heredity, as a result of evolution, in the case of objects desirable for the health of the body; but in greater part to associations (see Association) with previous similar perceptions, and in greatest part to feelings of satisfaction or dissatisfaction with the result of one's actions.

Sensations of moderate intensity are usually pleasant; sensations of very great intensity which produce abnormally strong reactions in the nervous tissues, are usually unpleasant, while those which have a complicated ratio are usually also unpleasant, which is especially true for musical sounds. Foods which nourished our ancestors usually taste good to us.

The child possibly acquires a taste for sweet things from the sugar in its mother's milk. Most of our pleasant and unpleasant sensations are the result of our education. They are, therefore, much more pronounced in adults, especially educated ones, than they are in children; although the expression or manifestation of them is less pronounced, more inhibited or restrained by other cortical activities. A perception which is associated with, or followed by, pleasure or pain will always, or for a long time, as often as it occurs actually or in memory, be accompanied by a pleasant or painful emotion, whether the memory of the original pleasant or painful result associated with it is present in consciousness or not.

On the other hand, our emotions or rather "our moods," depend in great part on our success or failure in life. Mankind does not find itself in this world with all its needs and wants satisfied; on the contrary, everyone must acquire food, clothes, habitation, warmth and a hundred other necessities. A man who sees his neighbor with something good, which he has not, desires it, or something similar. These wants and desires are the great, almost the only, incentives to voluntary action. When this voluntary action results in success we have in it our greatest pleasure and when it results in failure, our greatest unhappiness. All things connected with our success receive an associated emotion of happiness; while those things connected with our failure receive an associated emotion of grief.

In these various ways a certain number of our perceptions have associated with them an emotion (204), or mood, or tone, of pleasure or pain, greater or less, and a series of such emotions, or one long continued, will make us happy or unhappy for a considerable length of time and will constitute what we call our "mood."

In certain abnormal states of the cerebral cortex (exhaustion, circulatory irregularities, poison and other less known disorders) the emotions become dissociated from the ideas with which they are normally associated; so that all cerebral activity is accompanied by one emotion: in some cases, sadness; in others, fear; in others, joy and in others apathy or absence of all emotion. An emotion is often so strong and so occupies the patient's consciousness that it is impossible, or nearly so, to attract his attention. Such an abnormal mental condition occurs in some forms of insanity.

Association

The essential physiological characteristics of nervous tissue are: first, its excitability, its reaction to stimulation by the discharge of nervous energy stored within it, and second, its transmissibility; this nervous energy, wherever produced, does not remain localized, but tends to pass along nerve fibers, throughout its own neurons and to other neurons. The channels along which it will pass depend upon the anatomical arrangement of the fibers. In consequence of heredity and evolution, certain channels are easier for the passing of this nervous impulse than are others. This is especially true of certain reflexes present at birth, such as breathing, sucking, etc. Other channels are made easy later in life by the constant passage of impulses along them. The more frequently an association fiber and the synapse connecting two fibers are used the better conductors they become. The way that has once been traversed and that has often been traversed becomes the easiest way. It is the way of least resistance and it is a universal rule, whether it be a foot-path or a conductor of electrical or of other force or a nerve fiber or cell or a synapse separating two cells, that the way of least resistance is the easiest way: the way usually followed. When not used for a long time, like a deserted garden path, the channel may be obliterated and the association lost.

When a perception occurs, impulses radiate out along the association fibers from that portion of the cortex which produces it. If at the same time another perception, (usually there are

many), or a vivid memory of a perception received a moment before, takes place in another portion of the cortex, the association fibers connecting these two or more portions of the cortex, where perceptions are occurring, or have just occurred, being acted upon at both ends, will convey impulses to and fro more readily than the other association fibers. The details of this process are obscure, but it seems evident that the longer and more frequently the association fibers are traversed by these impulses the better conductors do they become and these two perceptions become more and more easily excited the one from the other. They may also be excited through the mediation of a third memory associated with both of the others. The activity in the cortex does not long persist; so that when the associated idea is in consciousness, the original perception which awoke it is already, or soon will be, subconscious. Yet they are firmly associated together; so that whenever in the future one enters into activity it may excite the other. Thus, association between perceptions of the events and objects received simultaneously, or immediately before or after each other, are formed in a never-ending stream and the events and objects are considered as contemporaneous and often as related to each other. Subsequent experiences may verify and strengthen some of these associations and may disprove and unmake others. Associations with any one perception may be, and usually are, extremely numerous. There is also an association of words as well as of perceptions and the associations of words have no necessary relationship to the associations of the objects which they represent. Associations may be at first very imperfect and very difficult to form, but with repetition and practice become easy. The work of a child in school is difficult until by repetition he has learned thoroughly his lesson. Then the recitation is easy. Addition subtraction, etc., are at first performed slowly and with difficulty but later, in consequence of frequent repetition, rapidly and easily. The association between question and answer becomes firmly established.

When a number of perceptions are produced which are very similar and yet show more or less individual variations, as for instance perceptions of men or dogs, from a comparison of them and of memories of others, more or less similar, a *concept* or *idea* of a man or a dog is formed which includes all the individuals. From many examples of individual freedom of action, the abstract idea, or *abstraction*, of liberty is formed. A great many such abstract ideas are gradually formed and this process is facilitated by the use of language. But each idea is the result of experience: the result of a conglomeration or generalization of one or more perceptions and their associations, and, by the aid of language, is given a name. It has been said that "we can understand only so much of an abstraction as we know individual cases which sustain it." Thinking and reasoning are much simplified and made more rapid by the employment of these abstractions. A concept, idea, or abstraction, differs in different persons in consequence of their different experiences, of their education and of their associations with the perceptions involved.

Ethics

Inasmuch as the sensory and motor areas of the cortex are intimately connected together, some perceptions lead to voluntary action, which may result in pain, either directly as physical pain, or indirectly as mental pain, the result of punishment or condemnation; so that the action and the perception which led to it will become associated with these unpleasant sensations or perceptions, and these associated unpleasant sensations will tend to restrain further similar actions. Such acts, bringing with them a penalty, will be called wrong and there will gradually be formed a large number of associations which will be identified with the ideas of punishment and condemnation and which tend to prevent the performance of wrongful acts; just as another combination of associations which have become associated with pleasure, reward or praise, will be associated with good or right. A person's idea of what is right or wrong will depend upon his education, the result of experience and of teaching, and is the basis of emotions and ethics, and that ill-defined acquisition from teaching and experience, the so-called *conscience*, and may evolve into very elaborate and very controlling feelings and habits of thought. According as education has developed in a person one set of these associations rather than the other, a good or bad character, not from the person's own but from the community's standpoint, is formed.

These ethical ideas can of course be imparted from one person to another by language and, indeed, frequently are so imparted, but such ethical ideas are rarely so firm and convincing as those obtained from experience.

Concentration and Attention

It seems to be a general law in the physiology of the nervous system that when there is a strong activity in one part, the activity of the rest of the nervous system is inhibited. Thus, reflex activity can be inhibited by strong pain; and the reflex activity of the spinal cord is more or less inhibited when the brain is in activity. In the brain itself, when a portion of the cortex or a group of nerve cells is in activity, the activity of the other cortical areas. as well as that of the lower centers, is inhibited. The stronger the local activity, the greater and more extensive will be the general inhibition, and consequently the more this local activity will have a free and uninterrupted field. When an unusual or very vivid perception or idea is in consciousness it occupies the center of the stage. Consciousness consists of this one vivid idea and its associations; so that milder activities occurring in the cortex at the same time, which should produce, ordinarily, perceptions and associations, remain subconscious. This phenomenon is called concentration and is a very important function in nervous physiology. When the cause of this concentration is a perception, in addition to this inhibitory influence, impulses from the active sensory cortex radiate to the motor cortex and out to the perceiving organ and cause a change in its musculature in the form of greater tension, tonicity, change in its position, etc., which local changes heighten the power of the organ for the perception of stimuli. Concentration is only another name for attention and has been regarded as an effort, and an expression, of the will or will-power, but the primary and essential factor seems to be unusually strong cortical activity. Not infrequently the concentration is centered about an unpleasant idea, from the thought of which we vainly try to escape; yet it is forced upon our attention and we cannot free ourselves from it in spite of every effort of our so-called will. We are at times in a state of "expectant attention" in regard to some possible unpleasant perception, which state we cannot prevent, try as we may.

Reverie and Thought

The steady stream of perceptions originating from the excitation of the various sensory organs is constantly awakening associated memories, and these memories other associated memories, and so, while consciousness remains passive, (does not for the moment contain any strong or impelling idea) an ever-varying series of memories, visions, day dreams, etc., flow by. This is called a *reverie* or a *day dream*. But during this passive condition, some memory may be awakened which will arouse a number of associated memories (strong cortical activities) which will dominate consciousness and may pass to the motor area, producing action. This stronger cortical activity brings the faint or passive consciousness into a strong active condition of attention and the passive reverie will be converted into active thought and this latter condition is called the act of *thinking* or *reasoning*. The process of thinking is, thus, independent of speech; although speech is essential to its clear expression and certainly facilitates it, especially in its deep and profound forms. The question of attention is one which seems to require a more or less external will to keep the cerebral activity limited to one subject. Attention is, however, a manifestation of the activity of the association of ideas. If many associations at the same time bring into strong consciousness the desirability of investigating some one perception, this idea (desirability of investigation), which we may call "a" and which will have widespread associations, will bring into consciousness this perception to be investigated, which we may call "b" with its various associations. If one of these associations leads to others remote and unrelated, and away from "b" it will not go far before it will awaken some of the associations conected with "a," even the absence of "b" from consciousness will do this, and "a" will be brought into consciousness and through "a" the investigation will be brought back to "b" again. This action, like other nervous actions, grows stronger by use, just as a machine runs more smoothly and powerfully after its initial stiffness has been overcome by use; so a trained, educated, cerebral cortex, is able

to keep one set of memories present in consciousness (attention), to call up associated memories, to reject some, to keep others active and to compare them all together.

When we have forgotten a name, we often cannot by an effort of "will," however strong, recall it. The desire for the name starts series after series of associations in some way related to the name, which finally bring it into consciousness. Or the attempt may fail and the desire may be unsatisfied at the time. Hours or days afterwards the name may enter consciousness by some chance conscious or subconscious association and be recognized as the desired name.

Imagination: Creative Faculty

A perception usually calls up memories previously associated with itself or its memory. But unrelated memories may have previously been in consciousness simultaneously, or nearly so, and these memories may associate themselves with it and a combination of ideas and scenes may present themselves which are not the result of any unmodified previous experience. This is called *imagination*. Imagination is based upon memory. We can imagine nothing, the elements of which we have not previously perceived. But memory can present elements not previously associated together (men and wings) and these elements may combine themselves to produce a non-existing entity: *i. e.*, men with wings. Things may be so combined and modified as to produce an entity, strange, bizarre and never before perceived. The normal relations of parts may be altered out of all relationship to themselvs to produce something quite different from anything within our experience. Thus, things may be combined, which are the exact opposite of our experience, with very whimsical results. A new and not previously perceived possible grouping of well known elements may lead to experiments and discoveries of important facts, ideas, theories, etc. This is the creative "faculty" which shows itself actively in prose and poetic works of the imagination, in contradistinction to true facts or real history, and passively in day dreams. This result of cortical activity may in many persons be facilitated by the action of drugs and may in some cases be the cause of hallucinations and delusions, (as in hysteria, hypochondriasis and insanity). However strange the results of the imagination, the *elements* of these results (even of some of the most monstrous delusions) are former experiences.

Intelligence (Charts III)

Intelligence consists of the contents of object consciousness. It implies accurate formation of associations and their ready future availability. It is absolutely dependent upon memory, without which it cannot exist. The greater the number of memories and the more perfect and easier the recalling of associations, the greater is the intelligence. A person's memories depend primarily on perceptions derived from his sensory organs and on his experiences. The greater the number and the variety of his experiences the greater will be his intelligence, other things being equal. If any one sensory organ is absent or diseased from birth, memories of this sense will not be present and the intelligence will be diminished, unless this defect is in some way compensated for. Intelligence does not necessarily imply keenness of perception.

An increase of intelligence, though unusual, is not abnormal. In some cases this increase is due to a greater number of perceptions and ideas (the learned man); in some cases to better and wider associations throughout the entire sensory cortex (the wise man), and in some cases one portion of the cortex is functionally developed at the expense of others (the genius.)

A diminution of intelligence may be due to imperfect development, to impaired nutrition or to destructive lesions of the cortex. Perversions of intelligence (insanity), although they may, in part, be caused by peripheral lesions, are fundamentally due to disease or poisoning, or malnutrition of the cerebral cortex.

Personality (Charts III and XVI)

Personality is a term used to express all the energy and power which have been stored in the cerebral hemispheres by the activity of the cerebral cortex during the life-time of the individual. It consists of all his memories, sensations, perceptions, concepts, ideas, and their various associations,

which vary in character in different individuals. More especially, it consists of those strong associations which have become his habits and his habitual judgments and also of his emotions, and of all else that the cerebral cortex and its association fibers have accumulated within themselves in virtue of their activity resulting from the sensory excitations which it has received during the individual's experiences in his life. The possibility of this accumulation by the cortex and, to some extent, its character depend upon the hereditary structure and functional activity of the nervous system of the individual.

Personality is the sum of the conscious and subconscious content of the brain. It expends as this content grows larger and better systematized. It becomes greater as during life a larger stock of energy is accumulated in its associated memories. It is stronger as the consciousness is more intense (virility). The "Ego" is the result of a long series of experiences (former perceptions) by which the body is differentiated from the external world (corporeal ego), and by which the complex of memories and ideas which the cortex has accumulated is differentiated from that of other individuals and is peculiar to itself (mental ego).

The totality of one's memories constitutes his experiences. Many similar memories, or experiences, are gradually combined into a general idea or principle which becomes a guide, or association channel, for future judgments and actions and may persist after the individual memories or experiences upon which it was founded have been lost. Cortical excitations of this nature are followed at once by actions which are almost involuntary (habits) and are not the result of a balancing of many former memories and ideas (thought). In this way one's character or personality is built up. Ideas firmly fixed by tradition, education and habit, acquire an overwhelming emotional value. They not only exist in spite of experience but even mould experience into conformity with themselves. Personality is the result of the manifold working of natural forces. Had the natural forces been different the personality would have been different. Each personality has its own history founded on its own personal experiences. *A man's personality is not present at birth. It has been acquired or created during and by his life reactions in a brain whose organization and capacity have been determined by heredity.*

Personality, being founded on, and consisting of, personal experiences, is strongly individual; but inasmuch as the large majority of men in the same community have very much the same experiences, and as they discuss these experiences with each other, there springs up between them a friendly feeling as beings of the same nature and with the same interests, needs, desires and aims. According to its education from its experiences a personality may keep itself apart from others and strive only for its own well-being and may thus be selfish (an egotist), or it may merge itself into the social life of the community and strive for the well-being of its fellow men as well as its own and thus be generous (an altruist). In spite of much in common, each personality differs from others. Some by their educational experiences become contemplative men, others close observers, others men of action, etc. Some men are of weak character, who have always been indulged and have always followed the path of least resistance; some are of strong character, who have had to endure privation and have learned to control their desires. These different kinds of men cannot by any effort of will change suddenly their character, which has been formed slowly by countless past experiences, acting upon a brain the anatomical structure and physiological activity of which has been determined by heredity. The personality of a child has the potentiality of developing in the future, but the kind of development depends as much, if not more, upon the kind of future that is before it, as upon its heredity.

Personality seems to be the energy resulting from chemical changes which have taken place and are taking place in the cerebral cortex and to depend absolutely upon the integrity of the latter. When the cortex is exhausted, or diseased, personality may be changed under some unusual experience, resulting from the undue dominance of some local excitation of the cortex, either permanently or temporarily (double personality), or it may become completely lost (automatism).

There is no scientific or trustworthy evidence of the existence of any further factor in the form of any ethereal essence, a "mind" or "soul," as distinguished from what has been de-

scribed as "personality." Indeed this assumption rather complicates than simplifies the matter, inasmuch as it is contradictory to one of the most firmly established principles of natural philosophy: "the law of the censervation of energy." This law has not only been established by irrefutable proof in the inorganic world, but also has proved by experiment to be valid in animals and even in man. If the soul can produce, or stop, a cerebral activity of its own volition, thus creating or annihilating force which normally proceeds in an endless chain from one manifestation to another, then the law of the conservation of energy is no longer valid.

Mankind has been unwilling to allow that the casualties and laws which prevail in physical activities, can be potent also in the body and still less in the brain and "mind," because this controverts all their preconceived notions of the soul and its relation to God. The prejudices (pre-judgments) of most men will not permit them to regard the mental activities as the result of the physiological activity of the cerebral cortex, in the same way that the physiological activities of the internal organs of the body produce and cause the function of these organs. They are unwilling to regard psychology as identical with the physiology of the cerebral cortex.

In earlier historic times, the winds from the cardinal points of the compass, rippling streams, cascades, the waves of the sea, growing trees and shrubs, etc., (everything which exhibited motion), were each supposed to be animated by an indwelling spirit, by whom their motion was originated and maintained. At the present time, Naiads, Nereids, Dryads and other Nymphs, charming creatures of the imagination, have all been banished by the advance of knowledge. Only the indwelling, animating spirit of man, the soul, still survives in the belief of many.

If a needle, or bar of soft-iron, is rubbed with a magnet, or with a piece of magnetic iron ore, and suspended so that it can turn freely, it will turn towards the north. A philosopher in very ancient times, who saw this phenomenon, would probably have said that this iron bar contained an animating spirit that "willed" to turn to the north and when by any cause it was deflected it still willed and strove to turn again to the north. A modern scientist knows that a magnetized iron rod tends to put itself at right angles to the electric currents, which are always flowing around the earth, and hence it points north to the magnetic pole: one end of the axis around which the earth's electric currents revolve. He has proved these facts by many experiments and can prove them again at any time; although he cannot explain the final *cause* of the action of the earth's electric currents upon the needle. The "why," he does not know. He does, know, however, that it is inevitable, constant, and not the capricious will of an invisible and unproved spirit.

Whether an individual believes in a soul, or not, and explains the phenomena of life in accordance with this belief, depends upon his traditions, his education, his experiences and upon the personality which has been gradually created and developed during his lifetime by the combination of all those forces constituting consciousness and subconsciousness, and which personality may, in a sense, be likened to the older conception of the soul. It seems probable that consciousness, intelligence, personality, etc., areforms of energy and may, perhaps, be called spiritual in contradistinction to material. They certainly are absolutely dependent upon the blood supply of the cortex, and when this is arrested, personality with every other manifestation of consciousness ceases; but it still exists potentially and may be reanimated, if the circulation be restored after an interval of only a few minutes. If, however, the arrest of the circulation is so long that the cortex begins to degenerate or die, the personality is lost permanently, both actively and potentially.

Personality, is then, the energy of cortical activity accumulated during the life of the individual. It is the sum of all his perceptions, concepts, their associations in abstractions, ideas, moods, and knowledge and the demonstration of all these in manner and method of action. It is the so-called mental content of the individual, or better perhaps, it is the individual, since the functioning of all his organs and his internal secretions are factors in the productions of his moods and are peculiar to him. It is the "Ego." As it is energy, not matter, it may be regarded as the mystic spirit called the "Soul," whose manifestations have been for ages the riddle of the science of psychology and upon whose assumed origin and destiny has been built the faith of religions.

Insanity (Charts III and XVI)

There is an abnormal as well as a normal cerebral activity. Whether we believe in the existence of a soul within the body or not, certainly insanity is no longer regarded as the possession of the body by an evil spirit (demoniacal possession). This was a well established belief for ages, but it has long since been abandoned and we now regard insanity as caused by abnormal cerebral action. The conception of the control by a spirit, which we have finally rejected as regards insanity, the vast majority of mankind still retains for the healthy body, possibly because most of the few men, who really think, have not studied cerebral physiology.

We have considered briefly the actions taking place in the sensory area of the normal, healthy, cerebral cortex. In an abnormal cortex these actions are deranged. Local disordered cortical function produces local paralysis, or apraxia, or convulsions, or even hallucinations; while general disordered function produces coma, neurasthenia or insanity. Abnormal structure, whether the alteration be slight or great, and consequent abnormal function, of the cerebral cortex may be either congenital or acquired. The congenital form may be manifest in early infancy or may become apparent at any stage of the individual's development, as he successively meets tasks which require more and more intellectual power, when it becomes evident that his intelligence and ethics fall below the commonly accepted standards of the race or community of which he is a member.

Insanity is a term applied to those forms of cerebral activity in which the intelligence, consciousness, emotions, personality, ethics, etc., one or all, are absent or abnormal. It manifests itself in various forms of cerebral disorder and may be divided into two classes: the organic (or structural) and the functional. When the brain is congenitally malformed or defective in consequence of disease, or injury, in the mother's womb, or at birth, its action is necessarily impaired and the manifestations of its activity are either absent or abnormal. Such cases are evidently of hereditary or, more accurately, of congenital, origin. If this congenital defect be extreme and the child cannot talk he is called an idiot (1083); if the defect be less extreme and the child can talk he is called an imbecile (1090): a purely arbitrary, but convenient, division.

Other cases of congenitally defective brain there are, which are capable of perceptions, memories, associations and consciousness; and of a certain degree of education. Some are even precocious children, but their education cannot be carried very far and at about the age of puberty they show abnormal cerebral responses. They show a difficulty in, or an impossibility of, acquiring the more complicated concepts of morality, altruism, etc. They cannot form wise and true judgments and they become criminals, moral imbeciles, etc. These cases are classed together under the term the feeble-minded (1094). All these groups of congenitally defective children constitute the class of *amentia* (1078—more or less complete absence of a mind which never existed; in contradistinction to all other forms of insanity: those in which intelligence already acquired, more or less perfectly, is lost, more or less completely, and which constitute the class of *dementia* (1079).

Education and training have much to do with the development of the activity of the cerebral cortex and consequently with the individual's intelligence and ethics. There are individuals who, partly in consequence of a defective brain and partly in consequence of defective training and education, do not have normal experiences and form a number of abnormal associations and ideas, especially ethical. Such individuals comprise the majority of criminals and cranks. Such cases bridge over the separation between the congenital and the acquired forms of insanity. It is possible that a person with a normal brain, who is isolated from his fellow-beings and receives no training or education, will be feeble-minded or even an imbecile.

In *amentia* the cerebral cortex is so structurally or functionally incapable that it cannot produce perceptions, or register memories, or form associations, except of the most rudimentary kind. Persons suffering from this condition, then, have no material for intelligence, consciousness or ethics and are either incapable of any, or of only rudimentary, speech. In *dementia* an originally fairly normal cortex, in consequence of organic or functional disease, can no longer

exhibit sufficient activity to bring previously acquired memories into consciousness or to reproduce formerly acquired associations or to manifest other formerly possible, cerebral activities.

The form of dementia (loss of previously acquired intelligence) most closely resembling amentia is adolescent insanity, or dementia precox (1098), which occurs in apparently normal youths between puberty and the age of 25 or 30 years. They lose their mental and physical activity, and exhibit marked mental deterioration and consequent abnormality. Some of these cases make a more or less complete improvement and are able to lead a fairly normal life of rather subdued intensity. Other cases grow worse and terminate in dementia and death.

Still other cases of insanity exhibit a fairly normal mentality until a fairly advanced age, at which time they exhibit delusions and erroneous judgments, from which they construct somewhat elaborate *systematized delusions*. A careful consideration of the history of such cases shows that even from youth they have exhibited a number of abnormal judgments. These cases are cases of *paranoia* (1116) and are incurable.

In addition to the above classes of cases, in which the dementia is usually due to a *congenitally* defective brain, is a class of cases due to organic *disease* (lesions) occurring in an apparently normal brain which may, however, have an hereditary tendency to cerebral disease. One of these forms of *acquired* insanity is *senile dementia* (1107), which is due to atrophy of the cerebral convolutions owing to imperfect blood supply, which is itself often due (secondarily) to atheromatous arteries and which is incurable. Another form of organic brain disease eventually causing dementia is *general paresis* (1106) due to a syphilitic meningo-encephalitis. Brain tumors and abscesses and meningitis, may also in rare cases give rise to symptoms of insanity, probably by causing local or general disturbances of the cerebral circulation.

These cases of acquired organic brain disease terminate in dementia and death. Dementia also occurs as a terminal symptom in alcoholic dementia (1103), epileptic dementia (1104), secondary dementia (1105), and in most other forms of insanity which have persisted for years. Exhaustion of the cerebral cortex from worry, anxiety, shock and other causes may cause insanity in persons with an unstable brain, as may also an anemic and altered condition of the blood. It is, of course, possible that several of the above causes act simultaneously, or in sequence, and as a matter of fact they frequently do so.

Many forms of insanity are not due to organic brain disease. No lesion can be found after death and they must, therefore, be classed among the *functional diseases* of the brain. Many of these are due to poisons (alcohol, germ toxins, ptomaines, etc.), others are due to mental strain and especially to all possible forms of worry, in persons with badly educated brains.

In most of these functional insanities, acute delirium (1110), delirium grave (1111), confusional insanity (1108), delirium tremens (1109), Korsakow's psychosis (1102), acute alcoholic mania (1112), simple delusional insanity (1113), symptomatic mania (1114), symptomatic melancholia (1115), the cardinal symptom is *delirium,* frequently associated also with an emotional disorder. In the other cases of this group there is a profound disorder of the emotions alone without any sufficient rational cause: mania (1118), melancholia (1117), circular insanity (1119), manic-depressive insanity (1120.)

We have already considered the emotions at some length. The emotions may at times attain such an intensity as to entirely dominate the personality and to profoundly influence both the thought and the activity of the patient and to produce clinical pictures of mania and melancholia described in Chart XVI.

In the functional insanities characterized by delirium we meet with a remarkable class of symptoms called illusions (214) and hallucinations (213), which are present usually in profusion. Illusions are always, and hallucinations are frequently, faulty and imperfect perceptions. Delusions due to faulty logic or faulty premises are common enough in the congenital and organic insanities; but not hallucinations and illusions, although they do occasionally occur.

An hallucination is much more vivid than a memory and is not a complete and correct reproduction of a former memory, but usually is something strange and bizarre. In some cases the hallucinations do not have the vividness of true perceptions, but seem to be internal voices or

suggestions, telephonic communications, or electrical action, etc. The process in the brain which produces an hallucination must be similar to that which produces a perception or memory, but the process is limited to the cerebral cortex, the spino-peripheral neurons playing no part in it, as they do in a perception. Hallucinations and illusions may occur as the result of a local disturbance in a brain which may not be for the moment entirely normal, although the individual is certainly not insane. In such cases, hallucinations and illusions can be quickly dispelled by reason and by proof of their abnormal character. Insane persons, however, in consequence of a diffuse cortical disturbance, cling to their hallucinations and illusions with great tenacity in spite of strong proof to the contrary. They cannot be corrected by the evidence of their other senses or by the evidence and reasoning of their friends. These hallucinations and illusions occurring in a brain weakened by nature, poison or disease, naturally lead to abnormal associations and consequently to abnormal ideas (delusions). Abnormal associations will result not only from the strength and vividness of these hallucinations, but also because from patches of meningitis and from patches of atrophy, or other cause, in the brain of an insane man some areas of the cortex have more blood and are more easily excited than others and, therefore, respond more readily to association impulses, near and remote. Moreover, these abnormal ideas, entering into consciousness and coming into conflict with former long established ideas, lead to a condition of consciousness which we call bewilderment, clouded, befogged, confusion, distrust, apprehension, fear, etc. In these cases impulses reaching the cortex normally from the organs of sense are so much weaker than the excitations already there, that they cannot enter into consciousness, but remain subconscious. They may, although subconscious, be registered and may be recalled to consciousness after the attack of insanity is past, but they have no present value and are inadequate to correct the abnormal activities and no sane judgment can result.

A cortex in which normal perceptions can occur only imperfectly, or not at all, and in which abnormal perceptions, associations and ideas are dominant, will naturally produce abnormal association reflexes, or actions. The simplest of these is *delirium* (217, 1109-10), in which the patient responds by word and act to the many false perceptions and ideas in his clouded and weakened consciousness. When the intensity of the process is less the false perceptions and ideas will produce *delusions* (215, 1097), which will cause abnormal and often dangerous association reflexes or acts. These delusions may remain isolated, unsystematized or may be woven in with all the real experiences of the individual's life; so that a systematized delusion, founded upon more or less evidence or reasoning, acting upon a weakened or limited general cortical activity, and one, therefore, incapable of producing a correct judgment, results.

Often in justifying or explaining a delusion a patient will give reasons or cite experiences, which we call false, but which are doubtless experiences, which have been presented to him by his abnormally acting cerebral cortex. These delusions, or false and uncorrectable judgments, naturally lead to acts which are incompatible with an unconstrained life in a reasonable community. Naturally, with all these abnormal cortical activities, not only the ethical ideas of the individual are changed, but also the normal emotions associated with normal cortical activity are profoundly altered, whether in the form of exaltation or depression, either continuously or in alternation with each other.

In all forms of insanity, in consequence of its abnormal content, consciousness is altered and personality may be changed. There may be a double personality or the individual may imagine that he is dead, an animal, a king, or God, or, in extreme degrees of dementia, the patient may show no consciousness or personality at all. The emotions are also altered (morbid temperaments) in accordance with ideas in consciousness, or may be entirely dissociated from the ideas with which they are normally in harmony, or may be entirely absent in extreme dementia, or may be feebly carried over from former highly emotional states. The emotion most frequently present, especially in the early stages of the disease is fear (phobia).

Fear and apprehension are prominent, even dominant, symptoms in the early stages of almost every case of insanity. The unusual, often monstrous, phenomena occurring in the cerebral cortex are so different from those previously present and so out of harmony with for-

mer memories and ideas, that the patients naturally become distrustful, apprehensive and full of fear. Many can hardly believe the information supplied by their own senses, much less the words of their friends. Some regard themselves as persecuted and as the victims of conspiracies. Fear is the dominant emotion within them.

The association reflexes are always altered in insanity in consequence of the abnormal cortical activity. In extreme dementia, voluntary motion is completely abolished. In profound melancholia, voluntary acts, as well as thought, are inhibited; while the reverse is true in mania, in which cortical activity, although abnormal, is greatly exaggerated. In consequence of prominent, compulsory ideas, so frequent in insanity, compulsory acts result.

Voluntary Action. Association Reflexes

The ultimate product of the complex mechanism of the nervous system is an action upon the animal's or man's environment for his benefit. We have already considered this activity in its barest outline under the title of "cortical reflexes." It seems desirable to consider these reflexes more fully in the light of our knowledge of the "psychic activities" and under the name by which they are generally known, "voluntary acts" or "association reflexes."

Activity never normally originates directly or spontaneously in the motor area of the cerebral cortex, but the cortical impulse initiating the movement always originates from the sensory area of the cortex. When a very strong excitation arises in this sensory cortex, as for instance, perceptions which are associated with the idea of imminent danger of death, this excitation passes to the motor cortex and thence down through the internal capsule and pyramidal tract and causes movements of flight and self-preservation. This act is as inevitable and as machine-like as is the simplest reflex act. A good swimmer bent on suicide cannot drown himself unless he is weighted or the action of his arms or legs is restricted. Escape from imminent death is for most men an imperative voluntary act. If, however, the danger is less great, as on a battlefield, the excitation leading to flight may still be there, but it may be inhibited by excitation from other associations, such as the idea of shame, love of country, etc., and the two excitations may neutralize each other. It is a question in any individual case whether the chemico-physical energies representing and causing the fear of death or those representing and causing love of country and honor are based on stronger experiences and wider and stronger associations. Whichever is the stronger prevails.

Ordinarily, when a perception, or memory, suggesting action has sufficient intensity to enter consciousness, the excitation is sufficiently strong to pass along the association fibers and awaken into activity the group of motor cells, called the "common paths," lying in the anterior central convolution and, if no other counteracting excitation comes to this latter portion of the cortex, the irritation passes through these great motor cells in the anterior central convolution and down through the internal capsule and pyramidal tract and the action takes place. Actions resulting from memories are usually weaker than those resulting from the original excitation or perception. When a number of more or less conflicting memories and ideas are in consciousness, some for and some against the action, impulses will be constantly coming to the motor cortex to be either immediately inhibited, or strengthened. The play of the different perceptions, memories and ideas, *the play of motives,* may continue a long time as the person *deliberates* and *exercises* his *free-will.* This merely means that the sensory activities causing the different memories, together with those causing the ideas derived from the mass of associated memories which constitute our ethics and those which constitute our emotions, have sufficient intensity to act upon the motor cortex, some as excitants, some as inhibitors. Fresh, allied memories constantly enter consciousness, because of their association with those already in it, and take part in this phenomenon. It is like a debating society in which arguments for and against are presented almost simultaneously, and the stronger argument rather than the will of the judge is the decisive factor. It is probable also that subconscious activities may play some part in this process and it is certain that the emotions exercise an almost dominant part in it. Eventually the stronger excitation will prevail and the act will be either done or left undone.

A voluntary act, depending upon, and being the result of, the association of ideas, may be described as an *association reflex*. The idea of the apparent freedom of will depends upon the absence of external compulsion and also upon the fact that the action takes place, or does not take place, in accordance with the relative strength of those cortical activities, which cause, also, our ideas and desires. The more perceptions and ideas a person has in his memory, the more learned and intelligent he is, the greater, wider and more protracted will be this "play of motives" and the more difficult will be the choice of the resulting action: the victory of any one set of motives. The very learned man is not the man of action. In a child, or in an ignorant man, with fewer elements of a choice, the association reflex will usually be more prompt. When a decision under the same or similar conditions is made a second time, and, especially, when frequently repeated, the association reflex takes place more and more promptly. In the frequent repetition of an act, practice, the same conducting channels and synapses are being constantly traversed and consequently become better conductors, and the act is easier to perform and is more or less automatic. It becomes a habit. A large part of our voluntary acts are habits. It is to be remembered, also, that the internal sensations and secretions which dominate our "moods," exercise a strong influence over voluntary movements which, under changing moods and altered internal sensations and secretions, may be very different at different times, although the causal external sensation is the same.

The gray matter, the point of union of the motor and sensory neurons, is in small compass in the subcortical centers and hence is well fitted for direct transference: for reflex and automatic actions. In the cortical centers, the gray matter is spread over a large surface and permits separate, local action, and consequently permits a large number of different memories and ideas, some positive and some negative, to act simultaneously upon the motor cortex and, thus, either cause or prevent a voluntary action. Both voluntary and reflex acts are for the benefit of the individual. The reflex acts depend upon heredity and evolution. They are the result of the experience of the individual's ancestors, of the experience of the race (phylogenetic). Voluntary acts depend upon the individual's personal experience (ontogenetic). The difference between the two depends mainly on difference in anatomical structure. In conditions which are new and in which no experience can guide him, an individual's voluntary acts are quite as likely to be detrimental as salutary. His reflex acts almost without exception are salutary.

The innervation memories stored in the motor cerebral cortex are originally acquired from reflex acts. The first voluntary acts of the child (sucking, opening and closing eyes, closing of hand, etc.) are adopted reflex acts, either unmodified or but slightly modified. A young infant does not will to suck milk from his mother's breast. It is a reflex act. But after the infant has experienced the result of this act a sufficient number of times, the sight of his mother, or hunger, may awaken his memory of this act and of the consequent relief from hunger, may awaken his *desire*, and he will suck the breast *voluntarily* in consequence of this active memory. Voluntary motions, or association reflexes, occur early and develop rapidly in infantile life, but occur much earlier, though they develop more slowly, in young animals. Chickens run almost as soon as they are born to their mother when she "clucks" for them. The lower animals thus possess at birth, by heredity, a more perfect nervous system; while human infants possess at birth by heredity, one less perfect, but capable of a wonderful development, which results in greater part from personal experiences.

The Play of Motives

The isolated cortical areas, containing the memories acquired by previous experiences, are all brought into activity by association fibers. When any sensory impulse requiring action is brought to the cerebral cortex, conflicting ideas, corresponding to those localized memories, must be brought into unison before any association reflex (voluntary action) can result. The cerebral activity may be compared to a town meeting.

At the town meeting a new condition presents itself for action. Shall some action be taken, or postponed, or refused? The subject is discussed from many angles; the result of memories stored in the sensory area of the cortex of the citizens. Many ideas acquired from former experiences

are advanced. The clergy bring forth certain *moral* factors, the result of a long education extending back to infancy when the brain was most impressionable and which have long been preserved in the brain as fixed principles. The business men advance certain *utilitarian* factors, experiences acquired through many business undertakings and which have been stored in the brain as fixed business principles and are emotionless. The poets and orators present the *emotions* of patriotism and glory which have been the result of a long education, which has stressed their vital importance for the community and each individual member of it. The shysters and rascals present *ideas of self-interest* and pecuniary expediency. Each view advanced calls up another, favorable or unfavorable. And so the dispute rages. The chairman, or moderator, takes no part in the discussion, while all these different ideas are poured in upon him. One by one inadvisable or impractical ideas die away. Their tendency to action is inhibited by the stronger impulse of the majority of other ideas. A greater unanimity is acquired by the remaining ideas. They pour in upon the chairman with ever greater intensity. He is set into activity and the overwhelming idea or impulse of the assembly for action pours through him to his Sheriff and Policemen: *executives* who correspond to the groups of motor nuclei which have been trained by previous reflex and voluntary acts to execute the "will," of the individual, which is really the dominant activity of his cerebral cortex.

Speech (Charts IVc and XIII)

The most complicated and important of all voluntary acts is speech. Although the lower animals can, in all probability, communicate with each other, speech and the allied functions, reading and writing, are peculiar to human beings and are the result of much instruction in the line of imitation and study. These functions, therefore, depend upon a healthy brain. If a child has such an imperfectly formed brain that he is an idiot (743), he consequently cannot speak. The perfection and content of speech, reading and writing depend upon education; being more imperfect the less the education and training and are, therefore, often quite abnormal, or even absent, in the defective and feeble-minded (752, 1090, 1094) and in hysteria (747-8) and insanity, especially in adolescent insanity (1098), in dementia (1079), in coma (745) and in insanity with diffuse cortical changes in the speech area (1106).

The power of speech is of enormous importance in the development of the race. It is the one factor which has enabled the human race to so far outstrip all other animals that it seems to form an entirely different order of beings from them. The spoken, and, still more, the written, word allows man to make his own all the experience, knowledge and wisdom of his ancestors and contemporaries, and raises him, thus, far above his own limited individual experiences.

Like all knowledge, the art of speaking, reading and writing is acquired from sensory impressions. The art of speech is not in-born, although its possibility, its mechanism, is. It is derived from the sense of hearing; so that when a child is born deaf, or acquires deafness in the first two or three years of life, he is also dumb: a deaf mute (744). A deaf mute can be taught to speak only very imperfectly, and then only by the sense of sight, or much more rarely by touch (Helen Keller). The art of reading and writing is derived partly from the sense of hearing and mainly from the sense of sight. If a child is born blind, or acquires blindness in the first few years of life, he can learn to read only books printed in a peculiar way, and then only by the sense of touch and hearing; the sense of touch replacing the sense of sight in these cases.

Many persons think that speech comes naturally, or by nature, to a child. Such is not the case. In a home and hospital for infants where the limited number of nurses were so busy in attending to the physical needs of the infants that they had little time for anything else, it was noted with some surprise that the children, grown to the age of eighteen months and two years, made no effort to speak. It was necessary to bring in some older girls for this purpose and to impress on the nurses the necessity of devoting attention to teaching children to speak. The first sounds which the child utters, little cooing sounds, are doubtless emotional reflexes, scarcely more complicated than laughing or crying. From these sounds, speech is gradually built up by careful training. The mother in her play with the child imitates these sounds and gradually changes them into so-called "baby talk:" into some resemblance to actual syllables or words. The

child who is continually being taught to imitate the mother, very slowly changes the simple cooing and other sounds into these nearly related ones and finally toward the end of the first, or during the second, year of life, Da-da is converted into pa-pa, and ma-ma, and other simple words. It is a play and a lesson which is taking place every day in every nursery in the land. It is a slow process, but after a time the child begins to utter words to which he at first attaches no significance or understanding, but to which he later attaches a more and more definite meaning. Thus, speech, like other activities, is gradually acquired from simple reflex acts.

During the first month of its life the child gives utterance only to vowel sounds, especially "oo" and "a." Toward the end of the second month he begins to utter consonants, especially, "m" and "t," followed in the third month by "b"and "g" and "r" and "n," and in the fifth month "k" is added. During the second half of the first year the child holds monologues, as if really talking. Some of the sounds can be represented by letters, others cannot. He shows a strong tendency to repeat the same sound over and over and over again, probably in part, because his vocabulary of sounds is not large. At the beginning of the second year the child is able to repeat with some accuracy the simplest sounds which he hears; but even when he learns to repeat, with some distinctness, simple words as "mama" and "papa" he attaches at first no meaning to them. At the same time, however, he is beginning to understand certain words that are said to him and in response to the question, "How big is the baby?" he raises his hands to approximately the level of the top of his head. Thus, at a certain stage of his development the child can speak words which he cannot understand and can understand words which he cannot speak. There is much doubt as to really how much he understands. The raising of his hands may be a conditioned reflex, similar to those already described, and he might raise his hand equally well to the words "How small is the baby?" or even the word "big" or "ig" or even to inflection of the voice without any spoken word at all. Thus speech is at first a modified reflex. After this process has gone on for a time the child acquires a great facility in imitating the sound of words, as he does in all kinds of imitation, and will often surprise his mother by uttering a sentence which he has never said before, although, doubtless, he has often heard it said by others. However doubtful may be his understanding of words in the early stages of this process, there is no doubt that this understanding is slowly acquired and finally it becomes evident that the child clearly associates an object with a definite sound, or word. He often points to an object, the name of which is spoken, or even leads one to it when he is, as yet, unable to walk alone.

It is evident that the perceptions and memories of spoken words are of fundamental importance in the art of speaking. These perceptions take place and these memories are stored, in right handed persons, in the posterior half of the left superior temporal convolution and in the posterior portion of the left island of Reil (Fig. 15); so that lesions of this area cause a profound disorder of speech: sensory aphasia (775-6). From this portion of the cortex impulses pass along association fibers (the fasciculus uncinatus) lying in the external capsule to the base of the left inferior frontal convolution (Fig. 15) and to the anterior portion of the island of Reil. A lesion in this region, also, causes a profound disorder of speech: motor aphasia (774.)

The distinction between motor and sensory aphasia is not always easily drawn. In some cases when a patient is unable to speak a desired word it may be very difficult to decide whether he has forgotten the innervation memories necessary to speak the word (motor aphasia —222). or has forgotten the word itself (sensory aphasia—223). In the latter case he may be able to repeat the word when he hears it spoken. Lesions of the external capsule, in which run the association fibers connecting the centers of sensory and motor speech (the fasciculus uncinatus), also, cause a profound disorder of speech (conduction aphasia).

Perceptions of written or printed words are formed and their memories are stored, in right handed persons, in the cortex of the left occipital lobe, and from this area impulses pass along the association fibers lying beneath the angular gyrus to the base of the left inferior frontal convolution and the base of the left middle frontal convolution, where are stored the innervation memories of speech and writing respectively. Therefore, deep lesions in the region of the left angular gyrus in right handed persons will cause a complete alexia (777) and an incomplete

agraphia (779). The area of the cortex in the left hemisphere described above, including the bases of the middle and the inferior frontal convolutions, the island of Reil, the posterior half of the superior temporal convolution and the angular gyrus, is called "the zone of language" and is the cortical center, or psychic center, for the faculty of language.

In addition to its cortical center, speech depends upon the integrity of the muscles and nerves which move the lips, tongue, soft palate, larynx and those concerned in respiration. In lesions of these muscles and nerves and of their nuclei in the medulla and pons and of the pyramidal tract, speech may be abolished (anarthria) or pronunciation impaired (dysarthria), whether in consequence of paralysis or of incoordination, or of spasm (as in stuttering). Reading and writing may be similarly abolished or impaired in lesions of the peripheral nerves or of their nuclei in the optic thalamus or in the anterior horns of the cervical enlargement of the cord or of the fasciculus of Gratiolet or of the pyramidal tract.

Dysarthria might also be due to a cortical paralysis of the pneumogastric nerve, but the laryngeal muscles have a bilateral cortical representation; so that if one cortical area be injured the corresponding area of the other hemisphere can carry on the function of speech perfectly. There is, therefore, no laryngeal paralysis, or consequent dysarthria, due to any lesion within the cerebral hemispheres, unless the lesion be very extensive and involves both hemispheres (pseudo-bulbar paralysis).

Automatic Movements (Charts III and XVI)

This term is applied to two quite different sorts of actions. In one sense automatic, or autochthonous, acts are reflex acts which originate, not from external, but from internal, or organic, excitations or irritations. One of the best examples of this activity is the respiratory act. Another is the gastric and intestinal peristalsis. Such acts are very numerous and carry on the nutritive activities of the body.

The name is also applied to voluntary acts which have been learned with more or less difficulty, but which have been enacted so often that they can be performed without consciousness. Such acts are walking, writing, piano-playing, smoking and many others which can be very perfectly performed unconsciously, although each one can be enacted consciously and usually is so done. The nervous impulses underlying these automatic acts, although they may be entirely unconscious acts, probably always pass through the cerebral cortex and are never shunted to the ganglia at the base of the brain. Otherwise it is hard to understand why in destructive lesions of the motor cortex, the corona radiata, or the internal capsule these automatic acts are abolished, as they surely are.

Involuntary and Abnormal Motor Responses

Spasms and Convulsions (Chart IVb). Spasms and convulsions consist in involuntary muscular contraction. They depend mainly upon irritation of the central gray matter, especially the cerebral cortex, and partly upon peripheral irritation.

Passive contracture and Thomsen's disease alone are purely of peripheral (muscular) origin. Many of the tonic spasms are reflex, some are the result of nerve root irritation (meningitis, tumors, etc.) and many are associated with degeneration of the pyramidal tracts.

The result of pathological and experimental investigation makes it evident that epileptic and epileptiform convulsions may originate from irritation of the motor cortex. When a slight, but lasting, local irritation of the motor cortex occurs, there results a local spasm, clonic and tonic, which extends from one extremity to another and finally becomes a general convulsion, accompanied in some cases by coma. When the irritation is stronger and especially when it affects both hemispheres there results first a tonic followed by a clonic convulsion and coma. Irritation of other parts of the cortex can also produce epileptic convulsions, if the irritation be strong enough and the motor cortex be intact. Tonic spasms, without clonic ones, may be obtained by irritation of many parts of the central nervous system. The epileptiform convulsion caused by cortical irritation may be accompanied by alterations in the cardiac action, in the respira-

tion and in the activity of other internal organs, as in the cases of ordinary epilepsy. Indeed, these changes in the cardiac action and in the circulation through the brain may be more essential factors than is the cortical irritation in the production of some of the symptoms of an epileptic attack.

The contractures which accompany cerebral paralyses are due to contraction of the stronger muscles, partly in efforts for voluntary movements from the brain, but mainly reflexly from the spinal cord.

The pathogenesis of many spasms and the localization of their origin, especially of the irregular spasm, are given in Chart IVb.

The various forms of spasms are at times quite difficult to recognize. It requires much experience to be able always to differentiate clonus, tics, athetoid and choreic spasms from one another and from the perversions of motion: tremor, ataxia and apraxia. This is unfortunate because the diagnosis by these diagnostic charts requires that the symptoms be correctly observed and named. The student should compare carefully what he sees with the definitions in the book and should observe as many cases as possible.

Ataxia, Apraxia and Tremor (Chart IVc).. When an impulse from a cortical motor center passes down to a group of nerve cells in the anterior horns of the spinal cord, it causes a definite synergic contraction of a number of muscles to produce the movements over which the group of nerve cells presides. As soon as this movement commences, a number of sensory impulses pass from the muscles and joints involved to the coordinating centers, especially to the cerebellum, and the movement is consequently coordinated and orderly. This coordination of movements is not inborn. It is acquired by experience and practice. The movements of a new born baby are always ataxic. When the function of the cortical center is impaired there results a paralysis or an *apraxia* (loss of skill) according to the degree of the impairment and when the coordinating apparatus is functionally impaired there results *ataxia*. In either case awkward, ill-adapted and uncertain movements result. In cerebellar lesions there is *asynergy*: the muscles taking part in the movement do not act together at the proper time and with the proper relative force to produce an orderly movement.

The motor apparatus, together with its sensory regulation, may be called the executive apparatus and it may be disordered in various ways:

1st. If the motor portion of this apparatus be injured there results a *paralysis* or *paresis*. See Chart IVa.

2nd. If the sensory or regulating apparatus be injured there results *ataxia*. See Chart IVc.

3rd. If what has been learned has been lost or impaired there results *apraxia* or *dyspraxia*. See Chart IVc.

Ataxia, incoordination of movement, always depends upon some disturbance of the sensory or regulating apparatus. It occurs in several distinct varieties, depending upon the portion of the sensory nervous system affected:

1st. Peripheral, or dynamic, due to lesion of the peripheral sensory neurons.

2nd. Cerebellar, or static, due to lesion of the cerebellum or its tracts, including the terminnation of the auditory nerve in the semi-circular canals.

3rd. Cerebral ataxia, to a lesion of the cerebral hemispheres.

1. *Peripheral*, or *dynamic, ataxia* (280, 644) is caused by an impairment or loss of the complicated sensations conveyed by sensory fibers from the muscles, joints and other tissues which is known by the name of muscle-joint sense (43 and 352). It affects all movements of the parts involved. It is associated with hypotonia (240), which allows an abnormal excursion in passive movements without the resistance normally offered under sudden stretching, and which may allow of abnormal positions of the extremities. The loss of the muscle-joint sense can to some extent be replaced by the sense of sight, which allows the patient to guide his movements by his eyes.

Cerebellar, or *static, ataxia* (281, 642) is caused by impairment of the function of the great coordinating organ: the cerebellum. It affects mainly, or only, walking and standing, which acts

resemble those of a drunken man, or become absolutely impossible. The sense of sight gives very little aid in such cases. Movements of the extremities while the patient is recumbent are fairly normal. With cerebellar ataxia is usually associated vertigo; although this latter symptom may not be pronounced.

Cerebral ataxia is due to a lesion of the sensory tracts and centers within the brain. If this lesion involves the sensory tracts in the medulla or pons or crura cerebri, the cerebellar tracts may also be involved and the ataxia may be either cerebellar or dynamic or both. In cases of cerebral hemianesthesia where the lesion is either in the optic thalamus, the internal capsule or the parietal cortex, the ataxia, which invariably results, is of the dynamic variety and is associated with hypotonia. Such cases of ataxia may be slight in degree and may show great and relatively rapid improvement. When the lesion is in the parietal cortex, the center for cutaneous and muscular sensibility, ataxia results, because of the loss of those sensations which are essential for the proper guidance of voluntary movements. In tumors of the frontal lobe, whether cortical or sub-cortical, ataxia is a common symptom and is of the cerebellar type; being doubtless due to involvement of the fronto-cerebellar tract. In cerebral ataxia, it is evident that the patient is trying to execute the movements and knows what he wants to do, but he executes them awkwardly.

Apraxia (282) may result from the loss of the purposeful idea which should prompt a given action. In lesions of the posterior central convolution or of the supra-marginal gyrus this idea cannot be formed (sensorial apraxia or agnosia), in which case the action which should follow the idea cannot originate; or when this idea is formed the memory is quickly lost (amnestic apraxia), in which case the appropriate action is begun, but never completed. In lesions of the anterior central convolution, or of the area immediately anterior to it, the purposeful idea may be present, but the innervation memories necessary for the production of the appropriate action are lost; so that the action cannot be performed (motor apraxia). When the association fibers connecting the anterior and posterior central convolutions are the seat of lesions, the appropriate action will not occur, or a somewhat similar action may be substituted for it (associative apraxia).

In any organized society much results from imitation and from instruction. Certain complexes of innervation feelings become by practice so firmly united, that what was at first done with difficulty and imperfectly, becomes easily and perfectly performed. These innervation complexes are not inborn (although their anatomical substructure may well be), but are learned and acquired by practice. These innervation complexes become memories (kinesthetic memories). Innervation memories may be conscious in early life when first learned, but may be unconscious later. Many of them may never enter consciousness. As long as these memories persist the corresponding action may be performed, consciously or unconsciously, as the final result of sensory impulses exciting them. Many complicated acts are not performed often enough to form an innervation complex, but must be performed consciously and with constant sensory guidance from many parts of the brain simultaneously (sight, muscle sense, touch, etc.).

Tremor (250) may be caused by rapid rhythmical interruptions of the innervation impulses passing to the muscles or by failure of a proper proportion or equilibrium in the innervation of the muscles and their antagonists, and is especially characteristic of lesions of the lenticular nucleus and of the red nucleus and rubro-spinal tract. Tremor usually ceases during sleep and is usually increased by mental excitement; although a very powerful emotion may arrest the tremor temporarily. It seems to be always of central origin. Clonic spasm from exaggerated reflexes must not be confounded with a coarse tremor.

Athetosis, or mobile spasm, is a slow twisting movement of the fingers and hands, either unilateral or bilateral. It often follows a hemiplegia or diplegia, and occurs most frequently in cerebral palsy of childhood. It is characteristic of lesions of the caudate nucleus and of the putamen.

Trophic Influences (Chart XVII)

The nervous system exercises an important trophic influence over many of the tissues of the body, in addition to influences over their blood supply through the vaso-motor system.

This trophic influence can be divided into two great divisions, motor and sensory. When the motor nerve cells of the central or peripheral motor neurons are degenerated or destroyed (as in lesions of the nerve fibers or of the motor cells, of which these nerve fibers are the axons), the nerve fibers springing from such degenerated cells undergo a rapid degeneration, as do also the muscles, in which these nerve fibers terminate; and in early life when there is motor paralysis, or immobility of parts of the body from any cause, these parts fail to grow normally.

When the sensory nerves are degenerated, as in syringomyelia, myelitis, tabes, lesions of the spinal ganglia or of the ganglia at the base of the brain, etc., in consequence of the anesthesia thereby produced, the body is no longer protected, by reflex and voluntary acts, from the many traumatisms to which it is frequently subjected and therefore ulcerations, arthropathies, ulcerations of the cornea and other trophic lesions result.

Some of the ductless glands, especially the pituitary and the thyroid, when hypertrophied or atrophied as regards their glandular structure, also produce widespread trophic disorders.

The Cerebro-Spinal Fluid (Charts VIII and XIX)

The central nervous organs (brain and spinal cord) are bathed in a fluid called the cerebro-spinal fluid. This fluid is secreted or transudes from the choroid plexus within the ventricles of the brain and thus may contain substances which are in the blood. It passes out of the ventricles at the inferior angle of the fourth ventricle, through the foramen of Magendie. If from any cause (tumor, meningitis, etc.) the foramen of Magendie is occluded, this fluid, constantly secreted, cannot escape from the ventricles and dilates these cavities more or less according as the sutures of the skull are ossified less or more completely; thus producing internal hydrocephalus. The cerebro-spinal fluid passing out of the foramen of Magendie becomes the subarachnoid fluid, which lies in the meshes of the tissue forming the deeper layers of the arachnoid. In this situation it can receive products of any inflammation of the meninges: albuminous substances (globulin) and cellular structures (leucocytes in acute, and lymphocytes in chronic, inflammations); so much so as to be cloudy or even purulent. The specific germs of the various forms of meningitis can often also be detected, as well as blood in hemorrhage and pus in abscess. In tertiary and quaternary syphilitic meningitis the Wassermann reaction is usually positive. The cerebro-spinal fluid leaves the cranial and vertebral cavities along the cranial and spinal nerves and through the Pacchionian bodies and enters the veins.

The cerebro-spinal fluid is obtained by lumbar puncture and the rapidity of its escape is evidence of the tension which it is under, which tension can more accurately be measured by a manometer. When the cerebro-spinal fluid is increased in amount, as in meningitis, or when a foreign body, as a tumor, is within the cranial or spinal cavity the tension of the fluid is usually increased. The examination of this fluid is, therefore, of much importance in disease of the cerebral and spinal meninges and in other intra-cranial and intra-spinal conditions.

Electricity and the Nervous System (Chart II)

Nervous conduction, although it has some analogies with electrical conduction, is due to an entirely different form of energy. But when nervous action takes place, whether in a peripheral nerve or in a central ganglion, there always occurs an electrical current through the nerve or ganglion in the opposite direction. So constant and delicate is this reaction, that it has been used to prove the presence of nervous activity. Moreover the electric current, both Galvanic and Faradic, can be conducted along nerve fibers, and changes in the tension of electricity so conducted in the nerve fibers cause contraction of the muscles in which they terminate, as is shown in Chart VII. The muscle fibers, also, respond directly to changes in intensity of a Galvanic current, but not to those of a Faradic current. Degeneration of a nerve can be shown by its reactions to electricity (Chart VIIb).

All forms of electrical energy are excitants for all the sensory organs, acting not so much upon the end-organs as upon the nerves themselves.

Other forms of electricity, especially static electricity and high frequency currents, are used as therapeutic measures but have no diagnostic value.

CHART I

Case-Taking

METHODS OF EXAMINATION OF PATIENTS SUFFERING FROM NERVOUS DISEASES.

Errors in diagnosis result more frequently from imperfect observation than from faulty reasoning.

	Data derived from	
Methods of Examining and Testing Patients	QUESTIONING	see chart I a.
	INSPECTION	see chart I b.
	PALPATION	see chart I c.
	PERCUSSION	
	ELECTRICITY	see chart I d.
	LUMBAR AND BRAIN PUNCTURE	
	OPHTHALMOSCOPY	
	LARYNGOSCOPY	
	THERMOMETRY	

CHART Ia

Questioning

Comprising Numbers 1 to 18

(Note)—The examination of every patient, who is conscious and intelligent, begins with a history of his health and of that of his ancestors. This is an important source of information, although usually less so than are the results of the physical examination. The taking of a reliable clinical history is something of an art, but at best we are absolutely dependent upon the truthfulness of the patient, as we rarely have means to check his statements by information from other sources. It is important to put the patient at his ease and to gain his confidence. The patient is vitally interested in his own case and it is best to let him tell his own story of his illness in his own way, without interruption. He is eager to tell of his personal sufferings and often becomes impatient and irritable if interrupted by questions as to his hereditary and previous illness, which may better be asked later. When he has finished his own story is the time to question him about his illness, more especially and fully in regard to the organ probably affected, but also concerning the function of the other organs of the body. This done, he should be questioned as to his previous illnesses, occupations, etc., and finally as to any special prevailing illness in his ancestors or relatives. It is important to ask as few leading questions as possible. Questions in regard to personal habits and venereal diseases should only be asked when absolutely alone with the patient, and then in a manner which assumes that all men are guilty of indiscretions. During our taking of the clinical history we should have the patient under close observation and can thus form a good judgment as to his manner and general mental and physical characteristics.

Methods of Examination of Patients Suffering from Nervous Diseases

QUESTIONING

Methods of Testing

1
History of present illness.
(Chart II)

Allow the patient to tell the story of the illness without interruption (see note on preceding page). Then ascertain the exact date and manner of onset (sudden or slow, prodromata, etc.) and the exact sequence of symptoms. Inquire into all details which may concern the case (headache, pain, paresthesiae, vertigo, insomnia, mental condition, emotions, memory, special senses, paralyses, spasms, fits, disturbances of organic reflexes, loss of weight and strength, etc.), whether of recent or of old date. Seek for any possible cause (injury, poisons, drugs, infections, worry, mental or physical overstrain, shock, etc.). Be careful not to suggest answers to nervous people. Inquire into previous treatment and its effect.

2
Family and personal history.
(Chart II)

Ascertain the occurrence in the present, or a past, generation of the family of consanguineous marriages, of any nervous diseases, especially the neuroses (neuralgia, epilepsy, hysteria, insanity, suicide, drunkenness, etc.), or of syphilis or tuberculosis. Note patient's age, full address, race, his mental and physical development, his school life, injuries at birth, occupation, habits (alcohol, drugs, venery, masturbation, etc.), exhaustion, anxiety, worry, dwelling and previous illness, such as rickets, infectious diseases, chorea, fits, tuberculosis, syphilis (use discretion in this inquiry: ask of women, about sore throat, skin rashes, miscarriages, etc., and, for men, a good question is: "Of course, like all the rest of us you have had the clap. Have you ever had the pox or any sore on the genitals?") This may make confession easier. Ascertain the condition of other organs (cancer and tuberculosis).

Psychoanalysis is a part of the personal history. It is a minute and exhaustive inquiry into the patient's previous and present actions, motives and dreams, especially in regard to unhappy and repressive influences in the sexual life, of which the patient is scarcely, if at all, conscious and which have been repressed rather involuntarily than deliberately. The repressed idea may express and reveal itself (to a skilled investigator) in dreams, symbols, phobias, etc. Psychoanalysis and its concomitant treatment may produce in some cases beneficial results, but equally good results may follow other treatment and the method, fascinating though it be, seems to the author to be fraught with danger and is not recommended.

3
Consciousness.
(Charts III & XVI)

Patient may lie in a stupor and make little or no response to questions, noises, shaking, pin pricks, or strong sensory irritations of any kind. He may appreciate neither his surroundings, nor his acts, nor the time and place, nor his own individuality. He can remember, after recovery, nothing of what happened while he was unconscious. There are all possible grades in impairment of consciousness from complete coma to a slight lack of attention and an inability to collect one's thoughts. This can be learned by conversation.

4
Sanity.
(Charts III & XVI)

Patient's conversation and manner may show that his brain acts in an abnormal way and that he entertains abnormal perceptions and ideas (hallucinations, delusions, compulsory acts, ideas, etc.). Ascertain if a change has taken place in the patient's normal mental state, and when. Note whether patient is elated, active, loquacious; or dull, inattentive, sluggish, distracted, evasive, suspicious, and why. Some cases may require prolonged observation. At times irritating questions may be desirable, in order to excite the patient.

5 Intelligence. (Charts III, XIII & XVI)

In testing a patient's intelligence, we test his *general knowledge* by asking him to name the different days and the different months and by arithmetical, geographical, political and historical questions. His *power of observation* by showing him a number of things and asking him later to describe them. His *power of attention* by asking him to add a long column of figures or underscore a letter wherever it occurs in a page of print. His *power of comprehension* by asking him to explain something he has read or heard. His *association of ideas* by giving him a word and asking what other ideas it suggests to him. His *mental reaction time* by the time he takes to solve problems, or to name an object, the picture of which is shown to him. His *moral sense* by questions in ethics.

6 Memory and understanding. (Charts III, XIII & XVI)

An apparent defect in intelligence may be due to lack of attention, or may be shown, by further questioning, by having him repeat long phrases, execute verbal and written commands and name objects shown to him, to be due wholly, or in part, to a loss of memory; either general (amnesia), or local (aphasia), especially to a failure to understand what is said to him (sensory aphasia); while reason and judgment are normal. Test memory for remote, as well as for recent, occurrences. Test memory of statements made a few minutes previously, or of events of the day before, or of years before.

7 Emotions. (Charts III & XVI)

Patients may show by their conversation, if suitably guided, or by their manner, or by both, whether they are emotional or not. The emotional state of the patient and the mental characteristics discussed just above, can often best be learned from the statements of friends and relatives. Curious fears, the so-called "phobias," (235) are often present.

8 Speech. (Charts III, XIII & XVI)

Patients's speech may be entirely absent (anarthria) or altered and very defective, rational or irrational; there may be a limited vocabulary or use of the wrong word (aphasia), poor articulation (dysarthria), tremor in voice, monotonous, scanning speech, omissions of syllables and words, and may show individual peculiarities, all of which are to be noted. Speech is tested by interrogation and spontaneous (voluntary) speech. Test also patient's understanding of letters, words and phrases spoken to him, his executing spoken and written commands, his picking out objects named; and have patient name objects, give sequences, i. e., numbers, days of week, months, etc., and repeat catch phrases, as "Round the rough and rugged rock the ragged rascal ran," etc.

9 Reading. (Charts III, XIII & XVI)

Ask the patient to read *aloud,* even short sentences, words, or letters only If this be impossible can he read to himself? Can he recite, can he understand what he has read? Can he execute written commands?

10 Writing. (Charts III, XIII & XVI)

Ask the patient to write, spontaneously, from dictation and from copy. Have him write the names of objects shown him. Note any defect in the character of the writing or in the ideas expressed. Can he read and understand what he has written?

11 Stereognosis. (Charts III, VI & XXII)

Ask the patient to name objects placed in his hand, his eyes being shut, after excluding anesthesia. Even without feeling them all over and without moving them about in his fingers, a normal person should be able to recognize many objects (metals, cloth, etc.) merely laid against the skin of his hand, face, foot, lips, etc. Stereognosis may, therefore, in exceptional conditions, be tested, although less perfectly, in other parts than the hands.

12 Sight. (Charts VI & XIV)

Ask the patient to read small print or Jaeger's test type at reading distance (10 to 16 inches, according to age, refractive conditions, etc.) and Snellen's test letters at twenty feet. If patient cannot read the appropriate line at twenty feet the loss of vision is expressed by the number of feet from the chart at which he can read this line divided by twenty. Thus at ten feet the vision would be expressed by $\frac{10}{20}$ In great defect of vision the patient may be able to see only dimly the hand moved before his eye, or may only be able to distinguish between light and darkness. In testing suspected malingerers, who claim to be blind in one eye, while testing the sight with plain or low-power lenses, both eyes being open, a greatly over-correcting high-power lens should be surreptitiously placed before the normal eye. Then, if he can read the test-type, he must do so with the eye which he claims to be blind.

13 Achromatopsia. Color sense. (Chart VI)

Ask the patient to match different colored worsteds of various shades by day light. Color blindness may also be detected, and perhaps more accurately, in a dark room with a lantern with colored glass.

14 Field of vision for white and colors. (Hemianopia) (Charts VI & XIV)

Place the patient with back to the window or light and have him close his left eye and with his right gaze at the observer's left eye. Then let the observer move his hands about in a plane mid-way between himself and the patient; so that each should see the hand at the same instant as it comes into the field of vision. The observer can see if the patient's eye wanders from his own and recall it. Test left eye in same way. If any defect in field of vision is suspected, use a perimeter. With a perimeter not only the field of vision, but also, by using different colored papers, the color field can be mapped out. Normally the color field is largest for blue, then for yellow, orange, red, green, etc., in the order named. If this order is changed there is said to be an "inversion of the cloror fields" (851-2). Normally the lines limiting the different color fields, when charted, are everywhere separate from each other. If they touch or cross there is "interlacing of the color fields" or "dychromatopsia" (851-2).

15 Hearing and tinnitus aurium. (Charts VI & XIV)

The patient's hearing may be tested by voice, watch, or tuning fork. Be sure there is no wax in the ear. Ascertain whether he understands what is said to him, i. e., executes spoken commands. Galton's whistle should be used for testing high and low notes. Each ear should be tested separately. Bone conduction is tested by holding watch or tuning fork firmly on skull. Normally a tuning fork, which, held on mastoid ceases to be heard, can still be heard when held close to meatus (Rinne's test). Normally a vibrating tuning fork, held on center of forehead, is heard equally in both ears. If heard best in the deaf ear (positive) the lesion is in external or middle ear. If heard best in the normal ear (negative) the lesion is in inner ear or in auditory nerve (Weber's test). We also ask about ringing in ears (tinnitus aurium). In testing a malingerer, who claims to be deaf in one ear, place in his ears the ear-pieces of a stethoscope with long rubber, not metallic, tubes and speak into the bell (chest-piece) held some distance behind his back. While so speaking compress first one then the other tube a number of times. It will soon be evident whether he can hear with each ear, since he cannot tell which tube is open and which is shut.

16 Smell. (Charts VI & XIV)

Ask the patient to name from its odor any fragrant substance (such as asafoetida, cloves, peppermint, etc.) held for a moment beneath each nostril in turn, the other being closed. Ammonia and acetic acid should not be used in this test.

17 Taste. (Charts VI & XIV)

Ask the patient to point to the name on a printed card of the taste of a strongly bitter, sweet, salt or sour solution touched from a medicine dropper, or a camel's hair brush, to one side after the other of the protruded tongue. The tongue should be well washed between each test.

18 Sleep.

The amount of sleep which the patient gets in the twenty-four hours is always an important question. Insomnia (agrypnia) is present in many nervous diseases and is apt to be exaggerated by patients; so that their statements should be controlled, when possible, by those of the nurses or relatives. Many symptoms, especially fears, are worse at night: "Pavor nocturnus of children."

CHART Ib

Inspection (mainly)

Comprising Numbers 20 to 43.

Methods of Examination of Patients Suffering from Nervous Diseases

INSPECTION

Methods of Testing

20
Facial expression and general appearance and behavior.
(Charts XVI & XVII)

The expression of the patient's face indicates, in most cases, the degree of his intelligence and his emotional state (sad or gay or anxious), and also may suggest the presence of certain diseases and conditions; such as myxedema and cretinism ((116314). acromegaly (1183), scleroderma (1165), exophthalmic goitre (1193), paralysis agitans (677), myasthenia (554), nasal obstruction, atheroma of temporal arteries, notched teeth, hazy cornea, the saddle-back nose of syphilis, etc. His general appearance and behavior often indicate his power of self restraint (inhibitory power, breeding), or the existence of hallucinations (213) of sight, hearing, touch, or of compulsory acts (218).

21
Walk.
(Chart XIII)

The walk of the patient may suggest the presence of hemiplegia (254), paraplegia (257), local paralysis (259), ataxia (motor or cerebellar) (280-1), spasm (242), atony (252), paralysis agitans (677) and other tremors (250), pseudo-hypertrophic paralysis (500), hysterical paralysis (527), foot drop, (bilateral in multiple neuritis and lead palsy, unilateral in acute anterior poliomyelitis), weakness, exhaustion, etc.

22
Skull.
(Chart XVI)

The skull should be observed as to type (brachy- or dolicho-cephalic, round or long heads), size (microcephalic—small, macrocephalic—large), rickets (box shaped), general or local hydrocephalus (bulging—posterior or anterior), fontanelles and sutures, asymmetry, tumors, etc.

23
Vertebral column.
(Chart X)

The spinal column should be observed as to curvature (angular or lateral), scoliosis, kyphosis, spina bifida (occulto), deformity (dislocation), Pott's disease, tumor, tenderness (by palpation), etc.

24
Eye.
(Charts V, VI & XIV)

Note the existence of arcus senilis, the condition of pupils (unequal, anisocoria (341), miosis (340), mydriasis (339), and irregularity), the presence of keratitis or iritis, prominence of eyeballs, nystagmus, squint, ptosis, paralysis, etc.

25
Pupillary reflex to light.
(Charts V & XIV)

Note whether each pupil, the other eye being covered, dilates and contracts as the eye is alternately shaded by the hand and exposed to light, or an electric light is flashed into it; vision being constantly fixed upon some distant object. When a pupil contracts to light (direct reflex) the pupil of the other eye also contracts (consensual reflex). Naturally a pupillary reflex will not occur when the iris adherent to the lens (posterior synechia) as the result of a former iritis, which itself is often due to syphilis (in which disease pupillary reflexes are of much importance) or to rheumatism.

26
Hemiopic reflex.
Charts V & XIV

Note whether the pupil contracts as light is flashed on each half of the retina alternately. A ray of light collected by a lens should be used in this test. This reflex is difficult to obtain, and recent researches indicate that its existence is doubtful. It has been found, however, in a number of cases and verified by post-mortem findings in some of them.

27
Pupillary reflex to accommodation.
(Charts V & XIV)

Note whether the pupil dilates when the patient looks at a distant object and contracts when he looks at one so near his face as to require convergence of the eyes. This test can be made on a blind man by having him first converge his eyes and then make the axis of his eyes parallel, by imagining that he is looking at a near and then then at a distant object.

28
Pupillary reflex to pain.
Charts, V, XIV (330)

The pupils dilate when the patient suffers acute pain. Therefore, they dilate reflexly when the skin of the face is sharply pinched, or pricked with a pin, or irritated by electricity.

29
Double vision, diplopia.
(Charts VI & XIV)

Note which eye deviates, however slightly, from the direct axis of vision and which eye lags more or less on movement of eyeballs in following the moving finger. Place a colored glass before the affected eye, move a bright object (candle) throughout the field of vision and have the patient note the relative position of the two images. The colored image will of course be the one seen by the affected eye.

30
Secondary deviation of the sound eye.
(Chart XIV)

Hold a card close in front of the sound eye. Have the patient look at an object so held that the weakened muscle must be brought into action. The sound eye covered by the card will be observed to move too far and when the card is removed the sound eye will quickly move back into proper position.

31
Nystagmus.
(Charts IV & XII)

The oscillation of the eyeball which constitutes nystagmus is often plainly to be seen. Extreme deviation of the eyeballs in one direction or the other makes it more evident, and at times demonstrates a nystagmus not otherwise apparent. If present, nystagmus is usually recognized while making the two tests 29 and 30. It should not be confounded with the irregular jerky motion of a weakened ocular muscle attempting to move the eyeball.

32
Tremor.
(Charts IV & XII)

Note any tremor of lips, or other parts of the body. Note its frequency, amplitude, its relation to voluntary movements and whether it is associated with muscular rigidity. In testing for tremor, ask patient to hold arms extended before him or over his head with fingers spread and motionless; or observe him place his hand in his trousers' pocket.

33
Convulsion and spasm.
(Charts IV, XI & XII)

Note any convulsion (269), spasm (245-6), contracture (263-4), athetosis (271), choreiform movement (272), etc., which may be present. These various forms of spasm are often difficult to recognize and differentiate from each other.

34
Paralysis (motor).
(Charts IV, X & XIII)

Note any obvious paralysis, such as ptosis. Note the naso-labial fold and the height of the angle of the mouth on each side. While under close inspection, patient should be requested to execute every possible motion: i. e., wrinkle forehead (look upward, or open eyelids held closed by observer), frown, open and shut each eyelid, move eyeballs up and down and to either side (note whether upper eyelid follows eyeball well downwards), whistle, laugh, distend cheeks, raise upper lip and each angle of mouth, protrude tongue straight and move it in all directions, raise uvula in phonation, close jaws and move chin forwards and jaw laterally, contract strongly all muscles of face at once, move head backwards, forwards and towards each shoulder and shake it, bend body in all directions, raise arms vertically, raise shoulder, adduct and abduct arm, flex and extend elbow, wrist and each finger, spread fingers, adduct, abduct, flex and extend thumb, pronate and supinate forearm while elbow is flexed, stand on each leg, raise body on tiptoes, adduct and abduct thigh, flex and extend thigh, leg, foot and toes.

35 Paresis. (Charts IV, X & XIII)

Make strong resistance to above mentioned movements while patient is executing them: i. e., pull on eyelids, on one angle of mouth, resist movements of jaw, or of bending head, or body, or of flexing, extending, adducting and abducting joints, compare the strength of the paretic muscle with that of a similar healthy one, when possible, with its fellow of the opposite side of the body. For future comparison, etc., the strength of the paretic muscle can be registered by dynamometers, of which the most practical is the one for the hand grasp. Or sufficient weights may be placed on hand, foot or head to overcome the attempted movement. By these tests the degree of the paresis can be approximately measured.

36 Myasthenia. (Chart IV)

Note whether patient tires easily on repeated or continuous activity of any set of muscles.

37 Diadocokinesia. (Chart IV)

Note whether patient can alternately extend and flex joints quickly and repeatedly. Test especially rapid alternate supination and pronation.

38 Ankylosis.

Note whether any joint is rigid, so that it cannot be moved. Ascertain the cause of the rigidity, whether bony union, contracted muscle or contracted scar tissue (muscle, ligament, skin, etc.).

39 Contracture. (Charts IV & XI)

Note whether any muscle is contractured with consequent impaired motility of the joint and whether this contracture can be overcome by force, with or without etherization (active contracture), or not (passive contracture).

40 Muscle tone. (Charts IV & X)

Note whether muscles are firm or flabby, and whether or not resistance is offered to rapid motions of joints while the patient tries to avoid voluntary resistance. Normally there is slight resistance. In disease the resistance may be altogether absent (atonia), or weak (hypotonia), or strong (hypertonia).

41 Trophic lesions. (Chart XVII)

Note whether any muscle shows atrophy or hypertrophy, or fibrillary contractions, or if there is any arrested development or trophic lesions of other tissues (especially ulcers, herpes, glossy skin, abnormalities of nails, etc.).

42 Coordination (synergy). (Charts IX & XII)

Note whether complicated movements are executed in an orderly manner while the patient's eyes are closed. Ask patient to walk, touch point of nose with finger tip, pick up objects, write, touch knee with heel of other foot, hold foot steady in one position, trace a circle in the air with foot, walk backwards, sideways and along a line, stand on one foot alone or on both feet close together, either side by side or one in front of the other (Romberg's symptom), stand on tiptoes or on heels, stand on one foot and trace a circle on the floor with the toe of the other foot. All these tests should be made both with eyes open and shut.

43 Muscle and joint sense. Deep sensibility (bathyesthesia, kinesthesia). (Charts VI & XII)

Note whether patient, with his eyes shut, can tell whether his joints are flexed or extended, or can duplicate with one extremity the position in which his other is placed. Note whether he can estimate weights correctly or can grade by weight loaded balls correctly. Note whether he can locate his extremities in space. To test this, his eyes being shut, an extremity after being moved about is held in one position and he is told to turn his head and eyeballs so that when he opens his eyes he shall be looking directly at his thumb or great toe. When he opens his eyes it will be plain to see whether they are directed right or not.

CHART Ic

Palpation and Percussion

Comprising Numbers 45 to 68

Methods of Examination of Patients Suffering from Nervous Diseases

PALPATION AND PERCUSSION

Methods of Testing

45 Circulation and respiration. (Chart XVII)

Note the color of the skin, the pulsation of arteries in neck, the condition of the jugular veins and the frequency and regularity of respiration, especially Cheyne-Stoke's respiration (425), whether respiration be costal or abnormal, or the diaphragm be immobile, unilaterally or bilaterally.

46 Pulse. (Chart XVII)

Note pulse of patient as to frequency, volume, tension (best tested by tonometer or sphygmomanometer) and irregularity in rhythm and force.

47 Difficulties in sensory testing. (Chart VI)

The result of all sensory tests (and the same is true in regard to tests for many mental symptoms) depends upon the patient's truthfulness. Deception is always possible and even with the most truthful patients the tests require much time and the results are often contradictory, especially so, in excitable and in uneducated patients, who cannot fix their attention continuously. Nothing should be present to distract the patient's attention and his skin should be warm. In some nervous diseases the patient has occasional, spontaneous sensations which interfere with the tests. Most patients under the education of repeated tests become more acutely sensitive. On the other hand, tests too long continued tire the patient and give rise to contradictory results. It is to be remembered that the sensibility of the skin both for tactile and painful impressions varies greatly in different parts of the body and in different individuals.

48 Tactile sensibility. (Charts VI & XIV)

With the finger tip (or with a smaller and lighter object, such as the head of a pin, a camel's hair brush, a pledget of cotton, a hair, etc.), touch the patient's skin lightly, having told him to say "yes" every time he feels the slightest touch. Or the patient may describe figures (space sense) traced on his skin with ink (to prevent dispute or doubt). A pledget of cotton is better for accurate testing than is the finger tip or a pin, because with the cotton the pressure sense (49) is eliminated. Of course, during all sensory tests the patient's eyes must be closed or covered. In some cases of hysterical anesthesia, if the patient is told to say "no" when she does not feel the touch, she will say "no" only at the instant she is actually touched within the anesthetic (?) area; showing that sensation is not abolished, although it may well be abnormal (Janet's test). Tactile sensibility, or, more properly, "space sense," or "localizing sense," (53), may also be tested with the esthesiometer; a pair of blunt dividers, by which it is noted how far the points may be separated and yet be felt as one. This distance varies greatly in different parts of the body (at the point of tongue it is one m. m., at finger tips two m. m., along back and on upper part of arm and thigh it is sixty-five m. m. The distance is smaller transversely than longitudinally on the extremities. Neither this compass esthesiometer, nor Hering's esthesiometer gives more valuable results than the pin-head tests. When mapping out an anesthetic area commence in the anesthetic area and work towards the normal skin. Do the reverse in mapping out hyperesthesia; i. e., from normal skin to hyperesthetic area. The electro-cutaneous test can be more accurately measured, but is of little practical value.

49 Pressure sense. (Chart VI)	Note whether patient can estimate correctly the amount of pressure exercised by the finger pressed against the skin, or by weights laid upon it.
50 Painful sensibility. (Charts VI & XIV)	Note whether patient feels pain when pinched, or when skin is pricked by finger-nail, pin-point, or other sharp substance. Many instruments have been devised for measuring more or less accurately the intensity of the painful impression.
51 Retardation of conduction. (Chart VI)	Note whether the painful sensation is felt immediately upon, or some seconds after, the painful contact.
52 Persistence of sensation. (Chart VI)	Note whether the painful sensation persists a longer time, after the painful contact has ceased, than is normal.
53 Localization. (Chart VI)	Note whether the point of contact, tactile or painful, can be localized correctly by the patient either by description or by pointing; his eyes, of course, being shut or bandaged.
54 Double sensation and polyesthesia. (Chart VI)	Note whether a single or painful contact causes two (double sensation) or more, sensations (polyesthesia).
55 Temperature sense. (Chart VI)	Touch the skin at numerous points alternately with small test tubes, one filled with hot, the other with cold, water, or with hot and cold bodies (spoons) of the same size and form. Certain points of the skin are especially sensitive to heat; others to cold. It is well, therefore, to test for heat and cold separately.
56 Pallesthesia. (Chart VI)	Note whether the patient feels the vibration of a tuning fork (vibration sense) pressed so firmly on the skin that the vibration can be transmitted through the underlying bone (osseous sense).
57 Cutaneous reflexes. (Chart V)	Stroke or scratch, as softly as will suffice, with finger nail or head or point of pin, the skin of the sole of the foot (plantar and Babinski), or a buttock (gluteal), or the inner side of thigh (cremasteric), or the side of abdomen (umbilical), or the hypochondrium (epigastric), or interscapular region (interscapular), or stroke firmly along the postero-internal border of the tibia (Oppenheim's reflex) and note the resulting movement of the great toe. The muscle itself must be felt and watched in cases where the resulting contraction is too slight to move the part.
58 Mucous membrane reflexes. (Chart V)	Touch with finger, straw, brush, or probe, the cornea or conjunctiva (conjunctival), or mucous membrane of nose (nasal), or palate (uvular), or pharnyx (pharnygeal), and note the resulting movement.
59 Vaso-motor reflexes. (Chart V & XVII)	Note the pallor or redness of the skin, also rapid changes and flushings with or without irritation, such as scratching with a pin or finger-nail (dermographia).
60 Ankle-clonus. (Charts V & X)	With leg relaxed, semi-flexed and well supported, strike or press the sole of the foot quickly, firmly and continuously upwards and note whether the foot oscillates or not. This clonus occurs at times spontaneously when the toe and not the heel rests on the floor ("spinal epilepsy").

61 Knee-jerk. (Charts V & X)

While patient is sitting on a chair with legs crossed, or better on a table with legs hanging free, or is lying in bed on his back with knees flexed, strike the ligamentum patellae a sharp blow with the finger, edge of hand, book or percussion hammer and note whether the foot flies forward. The amplitude of the excursion of the foot is not alone a safe guide to infer increase of knee-jerk, but rather its vigor, its quickness, and the presence of two or three additional oscillations as the foot falls back again. Even a continuous oscillation, or clonus, occurs in some cases (the so-called "spinal epilepsy"). More common than this clonus is a simultaneous contraction of the adductors of the other thigh when the knee-jerk is exaggerated. In order to obtain this reflex the observer must make sure that the muscles of the legs are completely relaxed. The extensor femoris muscle must be observed and felt in those cases where the resulting contraction is too faint to move the leg. Knee-clonus may be obtained in suitable cases by grasping the patella from above and pulling it sharply downwards. In some cases of disease of the cerebellum, in testing the knee-jerk the leg swings backwards and forwards like a pendulum; the normal check restraint being absent (Pendular knee-jerk).

62 Achilles reflex. (Charts V & X)

While patient is kneeling in a chair with his feet projecting free, the tendo-Achillis should be strongly struck with a percussion hammer and the movement of plantar flexion noted. Where the patient cannot kneel the leg may be supported in any position which relaxes it and the tendo-Achillis struck.

63 Dorsal foot reflex. (Chart V)

When the dorsum of the foot is struck sharply over the 4th or 5th metatarsal bone, note the dorsal (normal) or plantar (pathological) flexion of toes (Mendel-Bechterew's reflex—321, 457).

64 Elbow and wrist reflexes (Chart V)

The arm being relaxed, well supported and semi-flexed at elbow the tendons at elbow or wrist are sharply struck.

65 The jaw reflex. (Chart V)

The patient's chin is firmly grasped with finger and thumb or a flat stick is placed in the patient's mouth resting on his lower teeth, the mouth being half open, and then the stick or the hand holding chin is struck sharply downward and the closure of the mouth noted.

66 Kernig's reflex. (Charts V & X.)

With thigh flexed at hip and leg flexed at knee, the patient either sitting or lying, the leg should be quickly extended at knee joint and a strong resistance to such extension noted, if present.

67 Mechanical irritability.

Strike the nerve or muscle sharply with the finger or percussion hammer or press the nerve trunk or its tender points.

68 Reinforcement.

The tendon, and to some extent the cutaneous, reflexes can be made stronger and can be often made to appear when apparently absent, by diverting the patient's attention in any way, usually by having him pull strongly on his clapsed hands, his eyes being turned to the ceiling or to a picture at the instant the reflex is tested (Jendrassik).

CHART Id

Electricity, Lumbar Puncture, Brain Puncture, Ophthalmoscopy, Thermometry, Caloric Reaction

Comprising Numbers 70 to 80

CHART II

Analysis of the Etiological Factors of the Case

Comprising Numbers 81 to 194

Analysis of the Etiological Factors of the Case

List of nervous and allied diseases likely to occur as the result of the etiological factors obtained from the history of the case.

- **81 Heredity, including consanguineous marriages in neuropathic families (Predisposing cause).**
  - **84 Inherited Diseases** — Organic Diseases
    - 101 Idiocy and Imbecility
    - 102 Spina Bifida and Meningocele
    - 103 Hereditary (Huntington's) Chorea
    - 104 Hereditary (Friedreich's) Ataxia
    - 105 Myatonia Congenita
    - 106 Myotonia Congenita (Thomsen's Disease)
    - 107 Muscular Dystrophies
    - 108 Syphilis of the Nervous System
    - 109 Dwarfs and Giants
  - **85 Inherited Tendencies** — Neuroses
    - 110 Insanity
    - 111 Epilepsy
    - 112 Hysteria
    - 113 Chorea
    - 114 Neurasthenia
    - 115 Neuralgia and Migraine
    - 116 Drunkenness (Alcoholism)
- **82 Personal Factors (Predisposing causes).**
  - **86 Age**
    - Infancy and Childhood
      - 117 Cerebral Palsy of Childhood
      - 118 Acute Anterior Poliomyelitis
      - 119 Meningitis (tuberculous, etc.)
      - 120 Hydrocephalus
      - 121 Tetany
      - And all the inherited diseases except 103
    - Childhood and Youth
      - 122 Caries of Spine and Compression Myelitis
      - 123 Meningitis (tuberculous, etc.)
      - 124 Hereditary Ataxia
      - 125 Glioma
      - 126 Chorea
      - 127 Epilepsy
      - 128 Muscular Dystrophies
      - 129 Hysteria
      - 130 Insanity
    - Adult — All other forms of Nervous Diseases and many of those above given
  - **87 Sex**
    - More common in women
      - 131 Hysteria
      - 132 Exophthalmic Goitre
      - 133 Neuroses
    - More common in men
      - 134 Locomotor Ataxia (Tabes)
      - 135 Paresis
      - 136 Injuries
      - 137 Organic Diseases
  - **88 Race**
    - Jewish & Latin — 138 Neuroses
    - Anglo-Saxon — 139 Organic Diseases
  - **89 Dwelling Place, Habitation**
    - Tropical
      - 140 Beri-Beri
      - 141 Leprous Neuritis
      - 142 Sleeping Sickness
    - Dampness — 143 Neuritis
  - **90 Occupation**
    - Overstrain — 144 Occupation Neuroses
    - Poisons — 145 Neuritis

83 Etiological Factors (Inciting causes)	91 Traumatism	Physical	146 Wounds and Injuries 147 Hemorrhage in Brain, Cord or Membrane 148 Meningitis 149 Myelitis 150 Disseminated Sclerosis 151 Neuritis 152 Tumors 153 Abscess
		Psychical, Acute and Chronic	154 Hysteria 155 Insanity 156 Neurasthenia 157 Traumatic Neuroses
	92 Poisons Toxic	Metallic	158 Arsenical Neuritis 159 Lead Palsy, Colic, etc. 160 Mercurial Tremor
		Alcoholic	161 Multiple Neuritis 162 Neurasthenia
		Tobacco, Tea or Coffee	163 Tremor 164 Neurasthenia
		Narcotic	165 Drug Poisoning; Acute or Chronic
	93 Infections	Germs and Toxines	166 Neuritis 167 Meningitis 168 Myelitis 169 Acute Anterior Poliomyelitis 170 Landry's Paralysis 171 Neuralgia 172 Tetanus 173 Hydrophobia 174 Abscess
	94 Syphilis	Tertiary Syphilis	175 Gumma 176 Meningitis Gummosa 177 Neuritis Syphilitica 178 Endarteritis Syphilitica
		Post-Syphilitic Infections	179 Locomotor Ataxia 180 General Paresis
	95 Exhaustion	From Illness, Overstrain, Worry	181 Neurasthenia 182 Hysteria
		From Venery and Masturbation	183 Neurasthenia
	96 Extension of Inflammation	Caries of Skull or Vertebrae	184 Cerebral or Spinal Abscess 185 Sinus Thrombosis 186 Meningitis 187 Myelitis 188 Neuritis
	97 Arterial Disease		189 Apoplexy (cerebral, spinal or meningeal) 189a Thrombosis, Cerebral and Spinal 190 Intermittent Claudication
	98 Metastasis from Other Organs		191 Tumors 192 Tuberculous and Suppurative Meningitis
	99 Disease of Other Organs	Bright's Disease	193 Uremia
		Diabetes Mellitus	194 Diabetic Coma and Neuritis
	100 Cold is a doubtful direct, but probably an auxiliary, etiological factor.		

CHART III

Disturbances of Mental Activity

Analysis of the Symptoms of the Case (Semeiology)

Definition, Significance and Relationship of the Symptoms of Disease.

200 Disturbances of Mental Activity. (More fully described and discussed in the introduction Page 5).

201
CONSCIOUSNESS
The appreciation of one's existence and individuality as separate from the rest of the universe (Subject consciousness). The content of consciousness is the sum of the present perceptions of the various sensations (Object consciousness), together with the memories of past perceptions and judgments (Experience) (Chart XVI).

202
INTELLIGENCE
The power of ascertaining facts and reasoning upon them. The power of discovering the relation of things and of acquiring knowledge (Chart XVI).

In disease, consciousness and intelligence may be either diminished or perverted as is set forth in Chart III a.

Neither intelligence nor consciousness is exaggerated or increased in disease, although the latter may be apparently so (Self-consciousness). In such cases, however, there is a concentration or limitation of consciousness rather than an increase of it; an exaltation of the subject, with a lowering of the object, consciousness.

203
MEMORY
The power of retaining in the mind and of recalling at will perceptions and ideas formerly received. The more striking the perception and the more frequently it is repeated or recalled, the better becomes its memory (Chart XIII).

204
EMOTIONS
An emotion is a state of consciousness accompanied by a feeling of pain, pleasure, fear, anger, wonder, scorn. etc. In health a person's emotion is usually in harmony with his environment, but in disease it may be quite independent of the environment (Chart XVI).

In disease, memory may be diminished in whole or in part, and the emotions may be either diminished or exaggerated as is set forth in Chart b.

Memory is never increased in disease, although certain memories may be accentuated and others lost; even all may be lost.

CHART IIIa

Disorders of Consciousness and Intelligence

Comprising Numbers 201 and 202, and 205 and 218

Analysis of the Symptoms of the Case (Semeiology)

CONSCIOUSNESS

		Diagnostic Symptoms	Definition	Significance
201 Consciousness	Diminished	205 Coma	The patient lies in a profound stupor from which he cannot be aroused by irritation of any sensory organ (eye, ear, skin, mucous membrane, etc.). No voluntary acts are performed and the reflexes are abolished or diminished, except the circulatory and respiratory, which are often, but not always, deranged. Patient is unable to swallow. Lips and cheeks puff out during expiration.	These three conditions are not always sharply differentiated, but may merge into each other. They are due to loss or diminution of brain function in consequence of pressure upon the brain or of circulatory disturbances in it, or of poisons, etc. They occur in traumatism, and in many organic diseases of the brain and its membranes and especially of its blood vessels; also when toxic substances (morphia, etc.) or toxins (fever) are in the blood; also in Bright's disease and diabetes mellitus. Rarely the condition is functional.
		206 Semi-coma or Stupor	The patient is apparently in a coma but by strong sensory irritation can be aroused to some manifestation of consciousness. No voluntary acts are performed, but the reflexes are usually present. Patient can swallow. Patient may lie apparently awake, but really unconscious, with a low muttering delirium (Coma vigil).	
		207 Dazed, Bewildered, Somnolence or Sopor	The patient lies in a deep sleep or moves about automatically. Can be rather easily aroused, but does not fully appreciate his surroundings. Can speak more or less intelligently.	
	Perverted	208 Erroneous personality	A mental condition in which a person imagines himself to be different from what he really is; sometimes an animal, sometimes a famous character in history, sometimes God, etc.	Occurs in insanity (functional).
		209 Double personality Dissociation of personality	At intervals the patient is in a sort of somnambulistic state and presents an abnormal consciousness and personality. His memory at times is said to change with his personality, in which case he remembers only occurrences in former similar conditions and not those of his normal state, and vice versa. This latter is a very rare and doubtful condition and offers much opportunity for deception, and in some cases of hysteria may well be suggested by the examining physician.	Occurs in hysteria and epilepsy (functional).
		210 Automatism Somnambulism	A person performs complicated and apparently intelligent acts, while suffering from loss, or great impairment, of consciousness, and retains little or no memory of the acts done.	Brain is probably anemic or exhausted, or the patient is under the influence of a great emotion (fright). Occurs in epilepsy, insanity, hypnotism, and rarely in hysteria (functional); not uncommon in childhood during sleep.

INTELLIGENCE

	Diagnostic Symptoms	Definition	Significance
202 INTELLIGENCE — DIMINISHED	211 Amentia	Absence or defect of intelligence, which is congenital or is acquired in infancy before the intelligence has developed.	Due to a malformed or diseased brain. Occurs in idiots, imbeciles and feeble-minded persons.
	212 Dementia	Absence or defect of intelligence, which is acquired in later life in a person previously intelligent.	Due to atrophy or functional failure or diminution of blood supply of cerebral cortex. Occurs in insanity and is often its terminal stage.
PERVERTED	213 Hallucinations	Vivid perceptions of sensations (visual, auditory, olfactory, tactile, painful, etc.) not directly dependent upon any external corresponding reality; a sensation without an external object. They may rarely occur in normal individuals and then may be recognized or proved to be false, but they are usually regarded as real and are then associated with defective judgment and mental impairment, and therefore cannot be corrected.	Due to disease of the cerebral cortex, whether functional, circulatory, toxic or organic. Usually symptoms of insanity, or of extreme degree of neurasthenia, are also present. In insanity these perversions of intelligence cannot be corrected by reason and demonstration, and in neurasthenia are only rarely and imperfectly so corrected.
	214 Illusions	Erroneous perceptions. A false interpretation of an actual sensation, which is really of a different nature from that which the patient believes it to be. Frequently occurs in rational persons, especially in those with defective terminal sensory organs. In such cases easily corrected.	
	215 Delusions	Erroneous judgments (often, but not always, dependent upon hallucinations) which can be corrected neither by reason, nor by the evidence of the senses and which are not in accord with universal human experience, and are the consequence of mental enfeeblement. Delusions are systematized or unsystematized according as they are supported or explained by more or less coherent reasoning, or not. The systematized delusions are of much more serious prognosis.	
	216 Hypochondriasis	Delusions of imaginary symptoms and illness formed on an insufficient basis of abnormal sensations, which cannot be corrected and are associated with much mental depression.	
	217 Delirium	Irrational talk, or acts, or both in persons with diminished consciousness. Probably due in most cases to hallucinations, illusions and mental confusion; consequently its irrationality may be in part only apparent. Often occurs in fevers.	
	218 Compulsory ideas and actions (275)	Certain thoughts or questions or doubts, which are forever in the patient's mind and cannot be removed. They may be of any nature. Patients are irresistibly compelled by an unknown force to do certain acts or to say certain words, usually quite trivial. Patients recognize the abnormal character of these ideas and acts and are made very unhappy by them, but are quite unable to prevent them.	

Methods for the detection of disorders of consciousness and intelligence are described in Chart Ia.

For further discussion of these symptoms and the diseases in which they occur see Chart XVI.

CHART IIIb

Disorders of Memory and Emotions

Comprising Numbers 203 and 204 and 220 to 237

Analysis of the Symptoms of the Case (Semeiology)

MEMORY

Diagnostic Symptoms	Definition	Significance
220 Amnesia	Inability to recall former perceptions and ideas. Loss of memory in general. May be more or less extensive. May affect memories of the immediate, or of the remote, past.	Functional or organic disease of the cerebral cortex, often anemia, sometimes the result of fright.
221 Agnosia	Inability, more or less complete, to recognize objects and acts, their uses and meanings, which were formerly well known; the sensory nerves and end-organs and projection fibers being normal. Such patients perform idiotic and insane acts and put formerly well known objects to absurd uses (urinate in water pitcher, etc.). It may be regarded as a local amnesia. Among the various forms of agnosia are: Asterognosis (230), Alexia (229), Agraphia (228) and the different forms of Sensory Aphasia (223-6).	Lesion of a cortical sensory center, or of the association fibers connected with that center.
222 Motor aphasia (aphemia).	Inability to express by words some idea in the patient's mind, although there is no paralysis of the vocal organs and the patient can usually express the idea by gesture. A loss of memory of how to speak (innervation memories), especially names. A limitation of the vocabulary.	Lesion in or near base of left inferior frontal convolution and anterior portion of left island of Reil in right-handed persons, and of the right side in left-handed persons.
223 Sensory or Auditory aphasia (word deafness)	Inability to understand (although not deaf) spoken words formerly intelligible. Loss of memory of words formerly heard. Hence inability to recognize them when spoken (233).	Lesion in or near posterior part of left superior temporal convolution and posterior portion of left island of Reil in right handed persons.
224 Optic aphasia	Inability to name objects, which the patient sees clearly, although he can name them after feeling them. Loss of visual memories (232).	Lesion of left occipital lobe or of association fibers from this lobe in right handed persons.
225 Mixed aphasia	A mixture of the three forms of aphasia just described.	Any one or a combination of the above lesions, or a lesion of the island of Reil, or of external capsule in right-handed persons, in whom the above lesions are always in the left cerebral hemisphere, or, in slight degree, may result from carelessness, or alcoholism, or a mild dementia.
226 Paraphasia (Jargon speech)	The use of a wrong word, or the omission of a word, or the placing of the right word in the wrong place, in speaking, with consequent incoherent speech. Jargon speech is an extreme degree of this.	
227 Paragraphia	The use of a wrong word, or the omission of a word, or the placing of the right word in the wrong place, in writing.	
228 Agraphia	Inability to express in writing the idea in the patient's mind, although he formerly could do so and his right arm and hand are not paralysed.	Lesion in the base of the middle left frontal convolution, cortical or subcortical.
229 Alexia (Word blindness)	Inability to read words patient could formerly read, although he sees them clearly and there is no paralysis of his vocal organs.	Sub-cortical lesion beneath left angular convolution in right-handed persons.

203 MEMORY DIMINISHED

Diagnostic Symptoms	Definition	Significance
230 Astereognosis	Inability to recognize objects by the sense of touch, although there is no anesthesia present in sufficient degree to prevent it.	Lesion in or near cortex, or sub-cortex, of contralateral posterior central convolution.
231 Apraxia (282)	Inability to execute a desired act. Loss of skill in executing acts, although there is no motor paralysis present. Loss of innervation memories necessary to perform these acts.	Cortical, or sub-cortical, lesion of motor area of contralateral hemisphere.
232 Psychic blindness	Inability to recognize well known objects or to comprehend familiar things by sight, although the patient is not blind. Loss of visual memories, optic aphasia (224).	Cortical, or sub-cortical, lesion of left occipital lobe, except in region of calcarine fissure.
233 Psychic deafness	Inability to recognize and comprehend well known words and sounds, although the patient is not deaf. Loss of auditory memories. Includes sensory aphasia (223).	Cortical, or sub-cortical, lesion of left superior temporal convolution in right-handed persons.

EMOTIONS

204 EMOTIONS		234 Sadness (Melancholia)	Without adequate cause the patient is depressed and unhappy. There is a great repression of mental and physical activity usually. He can be influenced little, if at all, by reason; difficult to get his attention.	Functional or circulatory disturbance of cerebral cortex, especially cerebral exhaustion. Occurs in neurasthenia and especially in insanity. Fears and apprehension seem to be the basic symptoms of many forms of incipient insanity (Mosher).
	EXAGGERATED	235 Fear (Phobias)	Without adequate cause the patient is in constant fear of an impending calamity, or has an unformulated fear. He has an unreasonable fear of the danger of contamination from filth, germs, etc., (mysophobia). He dreads to cross an open space (agoraphobia), or to enter a small room or confined space (claustrophobia), or fears a storm (astrophobia), or syphilis (syphilophobia), or ill-timed urination (urophobia), or everything (pantophobia), etc. Can be influenced little, if at all, by reason. Frequently has a more or less unconscious sexual basis.	
	DIMINISHED	236 Joy (Mania)	Without adequate cause the patient is exhilarated. There is great exuberance of mental and physical activity. Careless and destructive. Can be influenced little, if at all, by reason. Difficult to get his attention.	
		237 Apathy	Without adequate cause patient is in a dull, stuporous condition. No expression of physical or mental activity. An automaton, submitting passively to whatever is done to him.	

Methods for the detection of disorders of memory and emotion are described in Chart Ia. For further discussion of these symptoms and of the diseases in which they occur see Charts XIII and XVI.

CHART IV

Disorders of Voluntary Motion

ANALYSIS OF THE SYMPTOMS OF THE CASE (SEMEIOLOGY)
Definition, Significance and Relationship of the Symptoms of Disease.

240
DISORDERS OF VOLUNTARY MOTION

The power of executing movements by an effort of will is acquired in early life. The process is quite obscure, but seems to depend upon the existence of innervation memories of past acts, primarily reflex. Voluntary motion depends upon the integrity of the central motor neurons (461) and of the peripheral motor neurons (462). In disease the power of voluntary motion may be diminished, exaggerated or perverted.

MUSCULAR TONICITY

Closely connected with the power of voluntary and involuntary action is the fact that the muscles of a normal person are in a condition of constant, slight, but varying, contraction. This is called muscular tonicity or tone. It is really a reflex act caused and maintained by many slight irritations, and can be abolished by cutting the posterior nerve roots. Muscular tonicity is increased (hypertonia) in destructive lesions of the central motor neurons and in some functional disorders. It is diminished (hypotonia), or abolished (atonia). in destructive lesions of the peripheral motor or sensory neurons, in lesions of the cerebellum. in sleep and in narcosis.

241
DIMINUTION
also called
AKINESIS and
HYPOKINESIS

244
PARALYSIS
A condition in which the muscles cannot be contracted by the strongest effort of the will. As commonly used the term includes:
PARESIS
A condition in which the muscles can be contracted only feebly by the strongest effort of the will.

The conditions under which paralysis or paresis occurs are set forth in Chart IV a

242
EXAGGERATION
also called
HYPERKINESIS

245
TONIC SPASM
A continuous, involuntary, muscular contraction of longer or shorter duration (572).

246
CLONIC SPASM
More or less rhythmical alternations of involuntary, coarse, violent muscular contractions and relaxations. Must not be confounded with a coarse tremor (571).

247
IRREGULAR SPASM
Involuntary acts of various kinds (293-4, 573-4).

The conditions under which the various forms of spasm occur are set forth in Chart IV b

243
PERVERSION
also called
PARAKINESIS

248
ATAXIA
Disorderly movements due to loss of power of coordination (638). Asynergia (281-2). Associated with hypotonia (252). Dysmetria (289.)

249
LOSS OF SKILL,
APRAXIA
Awkwardness.

250
TREMOR
Involuntary rhythmical oscillation of some part of the body or of a muscle. Less powerful, more rapid and more rhythmical than a clonic spasm but similar in appearance, especially when coarse. Tremor may be slow (3 to 6 per second) or rapid (8 to 12 per second). It may be coarse or fine (639).

The conditions under which the various forms of perversion of motion occur are set forth in Chart IV c.

CHART IVa

Motor Paralysis

Comprising Numbers 244 and 251 to 260

Analysis of the Symptoms of the Case (Semeiology)

244 PARALYSIS { CHARACTER / EXTENT

MOTOR PARALYSIS

	Diagnostic Symptoms	Definition	Significance
PARALYSIS CHARACTER	251 Spastic, or hypertonic, paralysis. (473). (Figs. 24-6)	A paralysis in which the muscles show increased tone and offer much resistance to passive motion, especially rapid motion. The normal excursion of the joint is restricted. The muscles have their normal volume and under the microscope their fibers show a normal appearance. The electrical reaction of muscle and nerve is normal (394). The tendon reflexes are increased.	Destructive lesion of central motor neurons (461). It occurs in diseases of the brain or spinal cord, or may be functional. Rarely a reflex spasm (268), especially preputial irritation in children, or pain, may simulate this condition.
	252 Flaccid, or hypotonic, or atonic, or atrophic paralysis (472). (Figs. 24-6)	A paralysis in which the muscles have lost their tone and offer little or no resistance to passive motion, even when rapid. The joint has a normal or even increased excursion. The muscles exhibit a great and rapid atrophy, and under the microscope their fibers show a loss of their transverse striation and various forms of degeneration (fatty, hyaline, etc.). The electrical reaction of degeneration is present (397). When muscles are completely degenerated (402) passive contractures (263) may occur, in which stage the flaccidity and free excursion of the joint are lost. The tendon reflexes are abolished or diminished.	Destructive lesion of peripheral motor neurons (462). It occurs in disease of the muscles, peripheral nerves, anterior horns of cord, or motor nuclei in brain stem. It is never functional, but may be somewhat simulated by joint disease. Hypotonia without muscular paralysis or atrophy occurs in cerebellar lesions, tabes and other ataxic conditions (240).
	253 Myasthenic paralysis (554)	A rapid tiring of muscles upon exercise. A myasthenic reaction to electricity (399.) Muscles show small foci of small round cells.	A lesion of the muscles and often of the thymus gland.

MOTOR PARALYSIS (Continued)

	Diagnostic Symptoms	Definition	Significance
PARALYSIS — EXTENT	254 Hemiplegia (478-9) (Figs. 17-24)	A paralysis with exaggerated tendon reflexes of one lateral half of the body and extremities limited by the median line in front and behind. It is partial, if limited to arm and leg; complete, if arm, leg, tongue, palate and face are all involved. In some cases of hemiplegia there are slight weakness and exaggerated reflexes on the other side of the body also, especially in the leg. Symmetrical, bilateral muscles, which have a common function and a bilateral cortical innervation, are not paralysed; at most temporarily weakened. Such are the ocular, masticatory, laryngeal, respiratory, diaphragmatic, bladder, rectal, etc., muscles. In cerebral hemiplegia certain muscles are, in most cases, more completely paralysed than others. These "predilection muscles of Wernicke" are the trapezius, the external rotators and adductors of the upper arm, the triceps, the supinators and abductors of thumb, the extensors of the thigh, the flexors of the leg and the dorsal flexors of the foot.	A lesion of the contralateral central motor neurons (461). Hemiplegia is usually due to a cerebral lesion, but the partial form may be due to a spinal lesion, very rarely. Very rarely, there may be no lesion, except an extreme local anemia or edema of the brain as in nephritis (hemiplegia sine materia).
	255 Diplegia (478)	A double hemiplegia involving both sides. May be complete or partial and not infrequently is limited to the legs, or the face (facial diplegia), etc.	A lesion, usually but not always cortical, of the central motor neurons or basal nuclei on both sides.
	256 Crossed paralysis (537-42) (Hemiplegia alternans) (Figs. 20-1)	A paralysis of one or more homolateral cranial nerves and of the contralateral arm and leg.	Always due to a lesion involving the pyramidal tract with other structures in the brain stem (460); either in the medulla (hypoglossal hemiplegia alternans—1290--1), the pons (facial hemiplegia alternans—1292), or in the crus cerebri (motor oculi hemiplegia alternans—1293). The nuclei, or the neurons, peripheral or central, of the cranial nerves are involved below the decussation of their central neurons.
	257 Paraplegia (480) (Figs. 24-6)	A symmetrical paralysis of both sides of the body. Usually only involves the legs and lower part of body, but may involve the arms and even both sides of the face.	May occur in lesions of the muscles (dystrophies—477), or of the peripheral nerves (neuritis—488-9), or of the spinal cord, or brain stem, or even of the cerebral cortex (bilateral lesion). The distinction between paraplegia and diplegia (255) is not always sharply drawn. In general diplegia is applied to paralyses of cerebral origin, paraplegia to those of spinal or peripheral origin.
	258 Monoplegia (479) (Fig. 15)	A paralysis of one extremity only, or of one-half of the face only.	May be due to lesion of motor cerebral cortex, or of the motor nuclei, or of the peripheral nerves.
	259 Local paralysis (481) (Fig. 15)	A paralysis limited to one or more muscles of the face, eye, mouth, neck, body or extremities. Less than a whole extremity.	May be due to lesions of muscles or of peripheral nerves, or of spinal cord, or rarely of motor cerebral cortex, or functional.
	260 Aphonia (737-8)	Inability to produce vocal sounds. Absence of voice, but whispering is possible.	A variety of local paralysis. Laryngeal paralysis, organic or functional.

Methods for the detection of paralysis and paresis are described in Chart I b.

For further consideration of these symptoms and of the diseases in which they occur, see Chart X.

CHART IVb

Spasm

Comprising Numbers 245 to 247 and 263 to 276

Analysis of the Symptoms of the Case (Semeiology)

SPASM

	Diagnostic Symptoms	Definition	Significance
245 TONIC SPASM	263 Passive contracture (Figs. 24-6)	A continuous contraction of long duration in which the muscles, tendons and ligaments have become anatomically shortened and cannot be extended by force, even under etherization. The muscle fibers are degenerated, while the connective tissue of the muscle is hypertrophied and usually secondarily contracted, as in other newly formed connective, or scar, tissue.	Due to muscular lesions and to degeneration of the peripheral motor neurons (462).
	264 Active contracture (Figs. 15, 17, 24-6)	A continuous contraction lasting weeks, months, or years, which can be overcome by force, either with or without etherization. Muscles are in a normal condition of nutrition. Most common in the arms, or legs, or neck muscles (torticollis). The active contracture of a hemiplegia is usually that of flexion in the arm and of extension in the leg.	Active contractures occurring in hemiplegia affect the muscles not absolutely paralyzed. When the contracture is overcome by the application of a plaster of Paris splint, the muscles often show a surprising degree of voluntary motion when the splint is removed. These contractures depend in part, on attempts at voluntary movements and on associated movements, but in greater part on reflex action from sensory irritation; the inhibitory action of the brain being cut off by the lesion. They never occur in hemiplegia in tabetics and in any case can be relieved by section of the posterior nerve roots. Such contractures are always of very bad prognosis as to recovery from the hemiplegia.
	265 Myotonia (590-603)	An active contracture of brief duration but much longer than a convulsive tic. It may occur at the commencement of voluntary motion (Thomsen's disease, or myotonia congenita) or may be excited by cold (Eulenberg's disease, or paramyotonia congenita). It is frequent in meningitis and tetanus in which it takes many forms, viz: "retraction of head;" "trismus," strong closure of jaw; "opisthotonos," arching of body backwards; "pleurosthotonos," bending of body to one side; "emprosthotonos," arching of body forwards and "orthotonos," holding of body rigid and straight.	
	266 Rigidity	An active contracture of such mild degree that it does not prevent passive, or even voluntary, motion of the part, although rendering it difficult (paralysis agitans, etc.—677).	Active contracture is sometimes due to paralysis of antagonistic muscles or to muscle lesions.
	267 Convulsive tics (598)	A violent spasm of momentary duration. If rapidly repeated it must be classed under myoclonus (270 and 598 to 601). If painful it is called "tic douloureux" (599).	All tonic spasms (not including passive contracture) are due to a functional disorder, or are reflex (especially in children), or are due to irritation (chemical, sensory or vascular) of central motor neurons (461).
	268 Reflex spasm	A spasm, usually tonic, caused by irritation of some sensory tissue. It is especially common after injury. Many, if not all, of these reflex spasms may be, really, hysterical and can be cured by suggestion (415).	Painful cramps, especially in legs, of the nature of myotonia or tics, may be due to a deficiency of water in the system, and to cold.

SPASM (Concluded)

	Diagnostic Symptoms	Definition	Significance
246 CLONIC SPASM	269 Convulsion (571)	Violent clonic contractions of many, or of all the, muscles of the body.	Clonic spasms are usually due to irritation of the cerebral cortex, but may also result from very exaggerated reflexes (clonus).
	270 Myoclonus or convulsive tics	Successive clonic contractions of one or of a few adjacent muscles. Repeated convulsive tic. Most common in the face muscles (blepharospasm (598).	
247 IRREGULAR SPASMS	271 Athetosis or mobile spasm (574)	Slow, worm-like, rhythmical movements, often associated with transitory contractures (spasmus mobilis), of fingers and wrists and more rarely of toes and ankles. Hyperextension is the predominant action. Usually unilateral, but may be bilateral. Much more common in children than in adults. Muscles of the neck, face and of other parts of the body are not infrequently involved. It never occurs in completely paralysed muscles.	Lesion is usually in the caudate nucleus and putamen of contralateral hemisphere and not causing complete paralysis. May occur in diffuse cortical lesions.
	272 Choreic movements Chorea minor (573)	Rapid, irregular, coordinated but purposeless movements caused by contraction now of one group of muscles, now of another, throughout the body; bilateral or unilateral (hemichorea). Cease during sleep. They often render voluntary movements ataxic and are usually associated with a mild degree of paralysis of the muscles involved.	
	273 Chorea major or magna (629)	Patient performs involuntarily and uncontrolably a complicated and apparently purposeful movement. Also applied to a coarse tremor or violent oscillation of a part of the body.	Functional disorders, occurring in the neuroses and in insanity.
	274 Habit chorea (627)	Patient frequently performs involuntarily, and usually unconsciously, the same act. Usually a small act.	Choreiform movements, as well as athetosis and tremor, may occur in lesions of the corpus striatum.
	275 Compulsory acts (218)	Patient is compelled by some power within him, which he cannot understand or explain, to perform certain acts against his will.	
	276 Associated movements (Synkinesis)	Unintentional muscular contractions, occurring when movements are executed, or attempted in muscles not directly concerned in the movement attempted; often the corresponding muscles of the opposite side of the body, often those of the face. Such associated movements are Bell's phenomenon (434), Strümpell's tibialis phenomenon (435), Babinski's associated movements in unilateral paralysis (436).	In such cases movements often associated together, but which can be easily dissociated voluntarily in health, cannot be dissociated in disease which cuts off voluntary action.

Methods of detection of spasm are described in Chart I b.

For further discussion of these symptoms, and of the diseases in which they occur, see Charts XI and XII.

CHART IVc

Perversions of Motion
Ataxia, Loss of Skill, Tremor

Comprising Numbers 248 to 250 and 280 to 294

Analysis of the Symptoms of the Case Semeiology

ATAXIA—LOSS OF SKILL

[Ataxia is a disorder of one or more synergic units (simple asynergia), or of complex cerebellar synergia (integrative asynergia), or a disorder of the cerebral cortex (apraxia).]

	Diagnostic Symptoms	Definition	Significance
248 ATAXIA	280 Motor ataxia (644) (dynamic ataxia) (Figs. 24-6)	Voluntary movements are executed in an irregular and disorderly manner, which is due to a loss of the co-ordinating power. Rarely associated with decided vertigo.	Is due to a loss of muscle sense (43) (deep sensibility). May be due to lesions of peripheral sensory nerves, or of posterior columns of cord, or of brain stem, or of cerebral cortex posterior to fissure of Rolando, or may be toxic (alcohol), or functional.
	281 Cerebellar ataxia (642) (static ataxia) (Figs. 19-26)	Walking and standing are inco-ordinate, but other acts are not, or only slightly so. Patient executes simple movements of his legs fairly well when lying in bed, but in walking and standing he lacks synergy of the muscles and staggers and sways like a drunken man. *Asynergia major* usually associated with vertigo (392).	Is due to lack of muscular synergy (42) (asynergy). Due to lesion or functional disorder of the cerebellum or its tracts, including the direct cerebellar tract in brain stem or cord, or to tumors in frontal lobe of brain or to disease of ears or eyes, or to poisons (alcohol, etc.). In lesions of the cerebellar hemisphere the disorder may be transitory; in lesions of the worm it is more permanent.
249 LOSS OF SKILL	282 Apraxia (Fig. 15)	Inability, or difficulty, in performing a desired and accustomed act because of loss, or derangement, of the innervation memories concerned in that act (Motor aphasia, agraphia, etc.). The voluntary movements are awkward but not exactly ataxic. The patients seem to have lost their initiative to action. Sometimes a desired action is replaced by a different one (231). Loss of skill: *Asynergia minor.*	Lesion of the cerebral "common paths" or loss of innervation memories, general or partial, due to cortical or subcortical lesions of the anterior or posterior central, or supra-marginal convolutions, or to the association fibers connected with these convolutions, or to functional or anemic disorders of cerebral cortex. (See page 37).
	283 Anarthria (737)	Absence of speech. Speech may never have been acquired, as in idiocy, or may never have been acquired on account of deafness, or it may be voluntarily restrained for a purpose: or it may be more or less involuntarily restrained, as in insanity or hysteria (Mutism 744-7).	May be either functional or organic and, if the latter, may or may not be due to lesions in the peripheral organs of speech. If not, it is called pure motor aphasia or aphemia.
	284 Dysarthria (738)	Such difficulty in articulation that speech becomes indistinct and blurred, but is probably never so great as to cause complete anarthria (283).	Occur in lesions of the medulla and pons (bulbar paralysis, Figs. 21-3) and of the cranial nerves. Also in diphtheria, hydrophobia, myasthenia gravis, rarely in trichinosis and frequently in hysteria (globus hystericus). These symptoms are due to a combined disorder of synergic units and of integrative synergia, or to nuclear or cranial nerve paralysis, or are functional.
	285 Dysphagia	Difficulty in swallowing.	
	286 Dysmasesis (554)	Difficulty in mastication.	
	287 Astasia and Abasia (652 and 795)	Complete inability to stand or walk but legs can be moved freely, even strongly, when lying or sitting.	A delusion or auto-suggestion, which occurs in hysteria. May occur rarely in cerebellar lesions.
	288 Adiadocokinesia (37)	Difficulty in repeating a movement rapidly, especially supination.	Occurs in lesions of a cerebellar hemisphere, or is functional.
	289 Dysmetria	An exaggerated extension of the fingers just previous to grasping an *object.*	Due to disorder of the synergic units. (282).

TREMOR

Diagnostic Symptoms	Definition	Significance
290 Passive tremor (646 and 647)	Involuntary, rhythmical oscillation or trembling of a part which is otherwise at rest.	Functional. Occurs in paralysis agitans, weakness, etc.
291 Intention tremor (645)	An involuntary tremor which only occurs when a voluntary motion is made, or is willed and is about to be made.	Functional and organic. Occurs in neuroses and in organic diseases (disseminated sclerosis). It is due to dissociation of synergic units and, therefore, is asynergia rather than tremor. It is caused by lesions of the cerebellum, of the putamen and lenticular nucleus and of the red nucleus and rubro-spinal tract.
292 Nystagmus (640)	An involuntary trembling or oscillation of eyeball, usually horizontal, rarely vertical, very rarely rotatory. Increased, or only occurs, on voluntary motion of eyeball, especially on extreme deviation. The rapidity of the oscillations varies from 60 to 200 per minute; their amplitude from 2 to 4 millimeters. Nystagmus may be oscillatory when the motion in each direction is equally rapid, or rhythmic when it is quicker in one direction than in the other.	Ocurs espeially in lesions of the vestibular and other nuclei in the pons, Deiter's nucleus in the cerebellum, the posterior longitudinal bundle in the brain stem, in disturbances in the semi-circular canals, and in weakness of ocular muscles, and in lesion of ponto-cerebellar angle, also in the caloric reaction (79), and in cerebellar disease (80). It is due to dissociation of synergic units (asynergia.)
293 Fibrillary contraction or fibrillation (641)	An involuntary contraction of a bundle of fibers of a muscle of short duration. When many occur in adjacent bundles at short intervals, waves of contraction run over the muscle, but do not cause it to contract as a whole.	Degeneration of those multipolar nerve cells in the anterior horns of the spinal cord and brain stem of which the motor nerves supplying the muscle are the axons. Rarely occurs in traumatic neuroses.
294 Myokymia (697)	A fibrillary twitching of the muscles occurring in healthy persons.	Normal. Exhaustion. Following excessive muscular contraction or exposure to cold.

250 TREMOR

Methods of detection of perversions of motion are described in Chart I b.

For the further discussion of these symptoms and of the diseases in which they occur, see Chart XII.

CHART V

Reflex Activity

ANALYSIS OF THE SYMPTOMS OF THE CASE (SEMEIOLOGY)

Definition, Significance and Relationship of the Symptoms of Disease.

296 REFLEX ACTS

An involuntary movement caused by irritation of a sensory nerve or terminal organ. Although not the result of a conscious intention, yet these acts seem purposeful and usually tend towards the protection of the body. In order that a reflex act may take place there must be a comparatively healthy reflex arc, consisting of a motor nerve, a sensory nerve and some gray matter connecting the two; or, in other words, a motor neuron and a sensory neuron connected together directly or by a bridging neuron. Reflex acts are inhibited and modified by inhibitory impulses passing down from the brain along the so-called inhibitory fibers, which are also the central motor neurons: the pyramidal tract (472-4, 811). (Figs. 19, 24).

297 CUTANEOUS OR SUPERFICIAL REFLEXES

A reflex act which originates from an irritation of the skin (57).

298 MUCOUS MEMBRANE REFLEXES

A reflex act which originates from an irritation of a mucous membrane (58).

299 TENDON OR DEEP REFLEXES

A reflex act which originates from the sudden stretching of the fibers of a muscle (60-6).

300 ORGANIC REFLEXES

A reflex act affecting one of the viscera of the body, especially the bladder or rectum (1).

301 VASO-MOTOR REFLEXES

A reflex act affecting the arterioles (59).

The conditions in which reflex acts are disorordered are set forth in Chart V a.

302 PUPILLARY REFLEXES

A reflex act affecting the pupil (25-8).

The conditions in which the pupillary reflexes are disordered are set forth in Chart V b.

CHART Va

Cutaneous or Superficial Reflexes,
Mucous Membrane Reflexes,
Tendon or Deep Reflexes,
Organic Reflexes,
Vaso-Motor Reflexes

Comprising Numbers 303 to 328

Analysis of the Symptoms of the Case (Semeiology)

CUTANEOUS REFLEXES

Diagnostic Symptoms	Definition, Elicitation and Location of Reflex Centers.	Significance
303 Plantar	Plantar flexion of the toes associated with a contraction of the tensor fasciae femoris (Brissaud's reflex) when the sole of the foot is irritated. (1st and 2nd sacral segments.)	The abnormal reflexes, Babinski, Gordon and Oppenheim reflexes and ankle-clonus, always indicate disease of the central motor neurons (461), except in infants, in whom these reflexes (except ankle-clonus) may be present normally, and in some cases of hysteria, in which an imperfect ankle-clonus may rarely be obtained. The Babinski reflex is most reliable in a diagnostic sense. The Oppenheim reflex is sometimes present when the Babinski is absent and vice versa. Kernig's sign indicates meningitis or meningismus; it is an important but not certain, diagnostic sign (320).
304 Babinski's	Sluggish extensive dorsal flexion of the great toe when the sole of the foot is irritated.	
305 Gordon's	Dorsal flexion of the great toe when deep pressure is made through the calf muscle on the deep flexor muscles beneath; the leg being completely relaxed.	
306 Oppenheim's	Dorsal flexion of the great toe elicited by firm stroking with a hard object, or finger, just behind the postero-internal border of the tibia from above downwards; the leg being completely relaxed.	Alterations in the tendon reflexes are of very much greater diagnostic value than are those of the cutaneous (except the Babinski) reflexes, which are in many cases inconstant, probably because the cutaneous reflex impulses may possibly pass through the gray matter of the brain (cerebellum) as well as through a wide area of that of the spinal cord.
307 Gluteal	Contraction of the buttocks when the skin covering them is irritated. (4th and 5th lumbar segments.)	
307a Anal	Contraction of sphincter ani upon pin pricks of anus. (5th sacral segment.)	Diminution of reflexes is usually of little diagnostic value, but their abolition is of great value and may be due to a destructive lesion of any part of the reflex arc (a peripheral motor neuron, a peripheral sensory neuron, or a central bridging neuron). When there is a lesion of the peripheral motor neuron, atrophic motor paralysis is present in addition to the loss of the reflex. When there is a lesion of the peripheral sensory neuron there is usually a sensory paralysis (anesthesia, etc.) in addition to the loss of the reflex. Diminution or abolition of reflex activity (cutaneous or tendon) may occur, at least temporarily, in acute diseases or other forms of irritation of the central motor neurons; also in cases of shock, exhaustion, coma, narcotism and after epileptic fits, (except Babinski); also by will power and by voluntary movements of the muscles concerned; also (except Babinski) in recent cases of complete separation of the brain from the reflex centers in the spinal cord, and, rarely, from increased intracanial pressure, also frequently in fevers.
308 Cremasteric	Drawing up of the testicle when the inner side of the thigh is irritated. (1st to 3rd lumbar segments.)	
309 Umbilical	Sudden movement of umbilicus towards the side of abdomen irritated. (8th to 12th dorsal segments.)	
310 Epigastric	Sudden retraction of epigastrium when the hypochondrium is irritated. (7th to 9th dorsal segments.)	
311 Interscapular	Drawing inwards of the scapula when the skin of the interscapular space is irritated. (5th cervical to 1st dorsal segments.)	
312 Corneal or conjunctival	Closing of the eyelids when the cornea or conjunctiva is irritated. (5th to 7th cranial nuclei.)	

MUCOUS MEMBRANE AND TENDON REFLEXES.

Diagnostic Symptoms	Definition, Elicitation and Location of Reflex Centers.	Significance
313 Nasal	Sneezing when the nasal membrane is irritated. (5th to 10th cranial and upper cervical nuclei.)	The abolition of the knee-jerk is of great diagnostic importance. It is absent in tabes, neuritis (multiple and crural), acute anterior poliomyelitis involving the extensor cruris, Landry's paralysis, lesion of the cauda equina or of the lumbar enlargement, during an attack of family periodic paralysis, after an epileptic attack and in cases of muscular dystrophy involving the extensor cruris muscles. It is usually abolished in Friedreich's ataxia and combined sclerosis, except in the early stages when it may be increased. It may be absent in cerebral compression (tumor or meningitis) and in some cases of cerebellar disease, and may then be unilateral. It may be absent also in the conditions mentioned in the preceding paragraph.
314 Auditory-or-bicularis reflex.	When a loud noise is unexpectedly made immediately behind the patient, his orbicularis muscles contract. This reflex cannot be inhibited and may serve to detect simulated deafness.	
315 Uvular	Raising of the uvula in phonation or upon irritation of its mucous membrane. (9th to 10th cranial nuclei.)	
316 Pharyngeal	Retching or gagging when the pharynx is irritated. (9th to 10th cranial nuclei.)	
317 Ankle-clonus	Oscillation of the foot when the ball of foot is pressed quickly and continuously upwards. (5th lumbar and 1st sacral segments.)	Exaggeration of the reflexes may be due to a mild inflammation, or to any irritation, of any part of the reflex arc. Strychnine increases reflex activity by irritating the nerve cells in the anterior horns. More commonly, the reflexes are increased by any lesion of the central motor neurons, thus cutting off the normal inhibitory influence of the brain, and are then associated with paralysis of voluntary motion. The presence of ankle-clonus, the Babinski reflex and the dorsal foot reflex indicates a lesion of the pyramidal tract much more certainly than does an exaggerated knee-jerk, unless the latter is associated with an adductor contraction. Very commonly the reflexes are increased in functional diseases (hysteria) and in nervousness.
318 Achilles reflex	Sudden plantar flexion of foot when the tendo Achillis is sharply struck, patient kneeling. (1st and 2nd sacral segments.)	
319 Knee-jerk	Sudden extension of knee when the ligamentum patellae is sharply struck. When this reflex is exaggerated it is usually accompained by a contraction of the adductors of the opposite thigh, or even by knee-clonus (61). (2nd to 4th lumbar segments.)	
320 Kernig's sign	Resistance to sudden extension of the knee.	Innervation of the muscles not concerned in the reflex act and diverting the attention increases reflex activity (reinforcement, 68).
321 Dorsal foot reflex	Sudden plantar flexion of the toes when the dorsum of the foot over the 4th and 5th metatarsal bones is struck. (5th lumbar and 1st sacral segments.)	The paradoxical reflex is of no diagnostic importance. It consists in a contraction of the tibialis instead of the calf muscles when ankle-clonus is tested for; also of a contraction of the flexors instead of the extensors of the thigh when the knee-jerk is tested for.
322 Elbow and wrist reflexes	Sudden extension or flexion of elbow or wrist when the corresponding tendons are sharply struck. (5th to 7th cervical segments.)	In the dorsal foot-reflex (Mendel-Bechterew) normally there is either no reflex or a dorsal flexion of the toes, but in cases of pyramidal tract lesions a plantar flexion of the toes occurs.
323 Maxillary reflex	Sudden closure of jaw when it is sharply struck downwards. (5th cranial nucleus.)	

ORGANIC AND VASO-MOTOR REFLEXES.

Diagnostic Symptoms	Definition, Elicitation and Location of Reflex Centers.	Significance.
324 Bladder or vesical reflex.	The retention of urine in the bladder by the sphincter reflex, the expulsion of urine by the detrusor reflex and the synchronous relaxation of the sphincter. (Hypogastric sympathetic ganglia.)	Inability to void urine, or to retain it, is sometimes due to nervousness and sometimes to mechanical obstruction (enlarged prostate or stricture), but any other serious disturbances of the organic reflexes indicate organic disease of the nervous system. It never occurs in diseases limited to the peripheral nerves, except in lesions of the cauda equina, and rarely in cerebral disease. It is most common in spinal disease: sphincter paralysis with empty bladder and constant dribbling of urine in lesions of lumbar enlargement, and detrusor paralysis with distended bladder and often with dribbling of urine in lesions above the lumbar enlargement. (Fig. 28.)
325 Rectal reflex	Similar to that of the bladder. (Hemorrhoidal sympathetic ganglia.)	
326 Ischemic reflex	A sudden pallor of the skin following an irritation and limited to the area of irritation.	
327 Paralytic, hyperemic reflex (dermographia)	Congestion of the skin following the ischemia due to irritation (tâches cérèbrales and dermographia).	Vaso-motor disturbances cause a disturbance of the nutrition of the part. Diseases which result from. or are associated with, disturbances of the vaso-motor reflexes are discussed in Chart XVII.
328 Reflex of spinal Automatism (Marie) Defensive (Babinski)	By irritation of the skin or deeper tissues of the leg, more especially by firm lateral compression and powerful flexion of the foot and toes there results a flexion of all the joints of the leg and a withdrawal of it upwards.	Lesion of the pyramidal tract.

The methods of eliciting the various reflexes are described in Chart I c.
Diseases in which the reflexes are altered are discussed in Charts X, XIV, XVI, XVII.

CHART Vb

Pupillary Reflexes

Comprising Numbers 302 and 329 to 341.

PUPILLARY REFLEXES

302 PUPILLARY REFLEXES

Diagnostic Symptoms	Definition, Elicitation and Location of Reflex Centers	Significance
329 Pupillary reaction to light (25)	Pupil contracts when light is thrown on retina of the same eye (direct reflex), and when light is thrown on retina of opposite eye (consensual reflex), and dilates when retina is shaded from light (ciliary ganglion).	The direct pupillary reaction to light is abnormal in lesions of any part of the reflex arc (optic nerve, corpora quadrigemina, the Westphal-Edinger cell group of the motor oculi nucleus, third nerve and ciliary ganglion). If the lesion is in front of the optic chiasm, there will result blindness of the corresponding eye with loss of the direct, but preservation of the consensual reflex. If the lesion is back of the chiasm there will result a partial, never complete, loss of the field of vision of both eyes, and both the direct and the consensual pupillary reflexes will be preserved. If double lesions occur in the proximity of both corpora quadrigemina and total blindness results, both the direct and consensual reflexes are lost. If the double lesions are posterior to the corpora quadrigemina and bilateral hemianopia or total blindness results, and both the direct and consensual pupillary reflexes are preserved. Both these reflexes are absent in deep sleep, narcosis, shock, coma, epileptic, and occasionally in hysterical, attacks; also absent in tabes, in many cases of paresis and in rare cases of syphilis alone; absent also when the eye is under the influence of mydriatics or miotics. A careful study of the pupillary reflexes will serve to detect many cases of malingering.
330 Pupillary reaction to pain (28, 335)	Pupil dilates in pain, pinching, pin pricks, etc., of skin.	This reflex may be deranged in lesions of the cervical sympathetic ganglia of the same side.
331 Pupillary reaction to accommodation (27)	Pupil dilates when patient looks at a distant object and visual axes are parallel and contracts when patient looks at a near object and eyes converge.	The pupillary reaction to accommodation is absent (cycloplegia) in lesions of the third nerve, sometimes after diphtheria, occasionally in alcoholism and when the eye is under the influence of mydriatics or miotics, also in myopia and in cases of deficient convergence.
332 Argyll-Robertson phenomenon (437, 891)	Pupil does not respond to light, but does respond to efforts at accommodation.	The Argyll-Robertson phenomenon occurs in almost all cases of tabes and paresis (in many of these cases a degeneration of the posterior columns of the cord has been found at autopsy) and very rarely in cases of syphilis in which there are no manifestations of either tabes or paresis for years afterwards. The reverse of the Argyll-Robertson phenomenon, i. e., the preservation of the light reflex and the loss of the accommodation reflex, occurs occasionally in diphtheritic paralysis and has been found associated with syphilis, basal meningitis, tumors of corpora quadrigemina and myelitis. It is extremely rare.
333 Immobile pupil (545)	The pupil responds neither to light nor accommodation, but in some cases may still dilate slightly on irritation of cervical sympathetic.	Immobile pupil may occur in lesions of the optic nerve or tract or in its nucleus or in that of the third nerve or in the ciliary ganglion or its nerve. It may also be associated with ophthalmoplegia externa or interna or both. When it occurs alone it is due to lesion in the nucleus. Immobile pupil also occurs in tabes, in epilepsy, in some forms of hysteria, in fainting, and in katatonic stupor.
334 Hemiopic reflex (26)	Pupil contracts when light is thrown on the unparalysed half of retina, but does not contract when light is thrown on paralysed half.	The hemiopic reflex occurs only in lesions of the optic tract or geniculate bodies (homonymous hemianopia) or of the central part of the optic chiasm (bitemporal hemianopia). The existence of this reflex is disputed by many observers.

PUPILLARY REFLEXES (Concluded)

Diagnostic Symptoms	Definition, Elicitation and Location of Reflex Centers	Significance
335 Cilio-spinal reflex (465, 1191-2)	Pupil dilates when the skin of the neck on same side is irritated, (cervical sympathetic ganglion) or when cocaine is dropped in the eye.	The cilio-spinal pupillary reflex is absent in lesions of the cervical sympathetic, and in many lesions of the medulla and lower cervical and upper dorsal region of the spinal cord (cilio-spinal center—465). Hippus is usually associated with a general exaggeration of reflexes.
336 Hippus	When the eye is suddenly exposed to light, there occurs a series of alternate contractions and dilatations of the pupil, gradually growing less in degree.	Westphal's pupillary reaction occurs in some cases of tabes and in paresis. The paradoxical pupillary reflex is of no diagnostic significance. It has been observed in tabes and in paresis and is the result of fatigue.
337 Westphal's pupil reaction	When patient's eyelids are held forcibly apart and he attempts to close them he not only turns the eyeball upwards (Bell's phenomenon) but also the pupil contracts.	Mydriasis may be irritative or spasmodic, due to irritation of the cervical sympathetic ganglion or nerve; or may be paralytic, due to paralysis of the third cranial nerve or the ciliary ganglion; or may be due to both causes. It occurs in children, and on taking certain drugs (mydriatics). It occurs also from irritation of the cervical sympathetic *directly* by incipient lesions in the cervical enlargement of the spinal cord and its membranes, or by tumors in the neck, or by excess of carbonic acid in the blood as in dyspnoea and *indirectly* by strong emotions and especially by pain; also in paralysis of the sphincter pupillae (iridoplegia) from lesions, such as optic atrophy, glaucoma, lesions of the third nerve or ciliary ganglion, which break, or impair, the reflex arc, and which usually cause more or less diminution of vision and a deficient perception of light; also in coma, in cases of increased intra-cranial pressure, and in some other cerebral and meningeal lesions, especially in their later stages.
338 Paradoxical pupillary reflex	Pupil dilates instead of contracting upon exposure to light or upon efforts of accommodation.	
339 Mydriasis	Dilated pupils.	Miosis may be irritative or spasmodic, due to irritation of the third nerve or ciliary ganglion; or may be paralytic, due to paralysis of the cervical sympathetic ganglion or nerve, or may be due to both causes. It occurs in old age, in deep sleep, or on taking certain drugs (miotics); also from irritation of the third nucleus or nerve, as in meningitis in early stages and especially in hemorrhage into the pons; and from excessive use of accommodation, as in watchmakers, etc.; also from paralysis of the sympathetic in lesions of the neck and of the spinal cord (syringomyelia). It occurs often in tabes, paresis, iritis, irritation of cornea and, temporarily, after excision of the Gasserian ganglion.
340 Miosis	Contracted pupils.	
341 Unequal pupils or anisocoria	One pupil is larger than the other when the eyes are at rest.	Anisocoria occurs in many conditions and is of little or no diagnostic value.

The methods of eliciting the pupillary reflexes are described in Chart I b.
Diseases in which these reflexes are altered are discussed in Chart XIV.

CHART VI

Disorders of Sensation

ANALYSIS OF THE SYPMTOMS OF THE CASE (SEMEIOLOGY)

Definition, Significance and Relationship of the Symptoms of Disease.

344 DISORDERS OF SENSATION

The power of receiving perceptions of the external world and of the occurrences in our own body (the basis of all knowledge) is acquired early in life. The nature of the process is entirely unknown, but it rests upon the power of storing up memories and of recalling them. It depends upon the integrity of the central and peripheral sensory neurons (463-4), as well as upon that of the terminal sensory organs and of the cerebral cortex (47 to 56). This power may be diminished, or exaggerated, or perverted in various diseases.

345 DIMINUTION

Either no perception or an abnormally feeble one follows a sensory irritation adequate in health to cause a perception (806, 811).

346 EXAGGERATION

An unusually strong perception, as compared with health, follows any sensory irritation (807).

The condiitons under which sensation may be diminished or increased are set forth in Chart VI a.

347 PERVERSION

The occurrence or modification of a perception such as never occurs in health (930).

The conditions under which sensation is perverted are set forth in Chart VI b.

CHART VIa

Diminution and Exaggeration of Sensation

Comprising Numbers 345 and 346 and 348 to 372

Analysis of the Symptoms of the Case (Semeiology)

SENSATION

Diagnostic Symptoms	Definition	Significance
250 DIMINUTION		
348 Anesthesia (complete) or Hypesthesia (partial). (Superficial sensibility)	A loss, or diminution, of the normal sensibility to touch upon adequate irritation. Normal sensibility varies in acuteness in different parts of the body and in different individuals.	Diminution of sensibility may be due to disease of the terminal end-organs, or to a destructive lesion either of the peripheral sensory neurons (464), (in which case all forms of sensibility are abolished over an area usually coinciding with, but smaller than, the distribution of the peripheral nerve, and the reflex acts in the same part are also abolished); or of the sensory central neurons (463), (in which case frequently all forms of sensibility are not abolished, and the anesthetic area does not correspond to the area of distribution of a nerve, and the reflex acts in the part are not abolished). Sensibility is abolished in coma, narcosis and often, apparently only, in hysteria. A broad zone of analgesia and, more rarely, of anesthesia also, about the body occurs in locomotor ataxia: "tabetic cuirass." The anesthetic area may coincide with the distribution of a peripheral nerve or with that a nerve root (peripheral lesion); or with the distribution of several nerve roots (spinal lesion); or the area may involve one-half the body: called hemianesthesia (cerebral lesion and hysteria). Anesthesia of one side of the face and of the opposite arm and leg, "crossed hemianesthesia," occurs in lesions in the tegmentum of the pons and in the restiform body in the medulla. Anesthesia may involve some portion of the body supplied by small branches of many different nerves, such as a hand, a foot, a leg, a forearm, etc., and be sharply limited: "stocking and glove variety" (hysterical). (Fig. 33.)
349 Analgesia or Hypalgesia	A loss, or diminution, of the normal sensibility to pain, which in health varies in different individuals and in different parts of the body.	
350 Thermic Anesthesia or Hypesthesia	A loss, or diminution, of the sensibility to variations in temperature. This loss may be more marked for cold than for heat and vice versa.	
351 Loss of pressure sense	Inability to distinguish differences in the amount of pressure made on the skin.	
352 Loss of muscle and joint sense or Akinesthesia. (Deep sensibility)	Inability to tell how strongly a muscle is contracted, whether a joint is flexed or extended, or where an extremity is situated in space. A very complex sensation.	
353 Apallesthesia or loss of osseous sense or vibration sense.	Inability to feel the vibration of a tuning fork pressed firmly on the skin.	Analgesia, thermic anesthesia and apallesthesia may be due to lesion of the central gray matter, or of the antero-lateral ascending tract, of the cord (Fig. 26) or of the peripheral nerves or, very rarely, in hysteria. (Fig. 26.)
354 Astereognosis	Inability to recognize objects by the sense of touch; anesthesia not being present.	Astereognosis always indicates a lesion of the cerebral cortex. (Fig. 15.)
355 Deafness or Anakusia or Hypakusia	Loss, or diminution, of sense of hearing.	Anakusia, anosmia, ageusia and blindness, may be due to a lesion of the sensory terminal organ, of the sensory nerve or tract, or may be functional. But these symptoms may occur in so many conditions unconnected with the nervous system that they may have very little diagnostic value in nervous diseases.
356 Anosmia or Hyposmia	Loss, or diminution, of sense of smell.	
357 Ageusia or Hypogeusia	Loss, or diminution, of sense of taste.	

	Diagnostic Symptoms	Definition.	Significance
250 DIMINUTION (Continued)	358 Blindness or Anopsia or Amaurosis	Loss of vision.	Hemeralopia associated with a central scotoma for green and red is not uncommon in tobacco smokers. In them, when the pupil is dilated in a dim light, the healthy part of the retina can act. This condition is quite different from snow-blindness, where the retina is exhausted by too bright and too long continued light.
	359 Amblyopia	Decided impairment, but not complete loss, of vision, especially for colors in the early stages. Usually in such cases the field of vision is made small by the loss of more or less of its periphery or by scotomata.	Nyctalopia is at times associated with congenital retinitis pigmentosa, with cortical (peripheral) cataract and with other defects in the eye, and from exhaustion.
	360 Hemeralopia	A condition in which the patient sees better in a dim light than in a bright one (day blindness).	Hononymous hemianopia is due to a lesion of the optic tract posterior to the chiasm, of the geniculate bodies, the optic fasciculus or the median surface of the occipital lobe of the opposite side of the brain (lips of calcarine fissure). (Fig. 16)
	361 Nyctalopia	A condition in which the patient sees well in a bright light but is almost blind in a dim one (night blindness).	Bi-temporal hemianopia is due to a lesion of the central part of the optic chiasm. Nasal hemianopia is due to a lesion of the lateral margin of the optic chiasm. Bi-nasal hemianopia cannot result from one lesion.
	362 Hemianopia	Loss of one-half of the field of vision.	
	Homonymous	Loss of the same half in both fields.	
	Nasal	Loss of the nasal half in each or either field.	Tetartanopia is due to a lesion of the upper lip of the contralateral calcarine fissure if it be a lower quadrant of the field of vision and of the lower lip of this fissure if it be an upper quadrant: very rarely to a partial lesion of the geniculate bodies or optic fasciculus. (Fig. 16.)
	Bi-temporal	Loss of the temporal half in both fields. In almost all cases of hemianopia a limited area of central vision is preserved.	
	363 Tetartanopia or Quadrantic Hemianopia	Loss of an homonymous quadrant of both fields of vision.	Achromatopsia may be due to a congenital defect or to defective education or may be the early stage of a gradually developing blindness or amblyopia. Due to mild, not completely paralysing, lesions of any portion of the visual tract in the broad sense.
	364 Achromatopsia or color blindness. Hemichromatopsia	Inability to distinguish the different colors from each other either throughout the whole, or in one-half, the field of vision.	
	365 Dissociation of sensation	Loss of some forms of cutaneous sensibility (usually for pain and temperature) with preservation of others (tactile). (Figs. 24-7.)	Dissociation of sensation always indicates a lesion of the central gray matter (syringomyelia) or of the spino-thalamic tracts, or more rarely at the ponto-cerebellar angle of the pons at the level of the auditory nerve. It occurs associated with motor paralysis of the opposite side of the body in some cases of Brown-Séquard's paralysis.

	Diagnostic Symptoms	Definition	Significance
346 EXAGGERATION	366 Hyperesthesia	Increased tactile sensitiveness. An unusually slight touch can be perceived. A very rare and even doubtful condition. It is usually employed when a touch causes an unusually great, even painful sensation, where hyperalgesia or haphalgesia (380) would be a better term.	Exaggeration of sensibility of all kinds is usually functional. More rarely it is the result of an irritative, rather than a destructive, lesion of the central or peripheral sensory neurons. It occurs in strychnine poisoning and tetanus. Hyperesthesia occurs as a zone at the upper limit of the anesthesia in many spinal lesions, and on the same side of the body as is the lesion in Brown-Séquard's paralysis. It is usually associated with increased reflex activity.
	367 Hyperalgesia	Increased sensitiveness to pain.	
	368 Thermic Hyperesthesia or Hyperalgesia	Increased, even painful, sensitiveness to heat or cold, or both.	Photophobia is functional, or due to eye strain, or to inflammation of some part of the eye, or optic nerve, or cerebral meninges.
	369 Hyperosmia	Increased, even painful, sensitiveness to odors.	
	370 Hypergeusia	Increased and unpleasant sensitiveness to taste.	
	371 Photophobia	Increased and painful sensitiveness to light.	Hyperakusia is functional, or due to ear diseases affecting the labyrinth, or to cerebral conditions causing hyperemia of the labyrinth (meningitis, encephalitis, tumors, etc.) and to spinal affections.
	372 Hyperakusia	Increased, even painful, sensitiveness to sounds.	

Methods for the detection of these conditions are described in Chart I c.
Diseases in which these conditions occur are discussed in Chart XIV.

CHART VIb

Perversions of Sensation

Comprising Numbers 347 and 374 to 392

Analysis of the Symptoms of the Case (Semeiology)

SENSATION

Diagnostic Symptoms	Definition	Significance
347 PERVERSIONS		
374 Pain (Figs. 33, 38)	Is an unpleasant sensation not felt in perfect health, except in cases of injury. It varies greatly in intensity. It presents different qualities, such as: tearing, cutting, burning, throbbing, darting, etc. It may be diffuse, or felt in a small area(localized),or may run along a nerve trunk (radiating), or may run half way or entirely about the body or an extremity (girdle), or it may be felt in an area which is itself anesthetic (anesthesia dolorosa.) Pains may vary as to time of occurrence, some showing a distinct periodicity (malaria, neuralgia and migraine), some occur at menstrual epochs. Some headaches occur in morning (uremic), others in afternoon (ocular) and others towards evening and at night (syphilitic). Some pains are increased by pressure (neuritis and neuralgia) while some are diminished by it (lead colic).	Perversions of sensibility, especially pain and paresthesiae, are often functional and are often due to irritation (pressure, chemical, inflammatory, etc.) of central or peripheral sensory neurons. Radiating and girdle pains are usually due to lesions of the nerve roots. Anesthesia dolorosa is due to a lesion of the central end of a sensory neuron which has been destroyed below this point and therefore can conduct no sensations from below up to the brain. Although pain may be felt as peripheral it may be of central origin and due to lesions of central neurons within the brain or cord. On the other hand pains due to lesions in the abdominal viscera may be referred to remote parts of the body or the head (referred pains—953).
375 Paresthesiae	Curious sensations rarely felt in perfect health, usually unpleasant but not severe enough to be called pain. They are numbness, tingling, formication, heat, cold, heaviness, tired feeling, hunger, etc.	Failure of localization may be functional but usually results from lesions of the peripheral sensory neurons (tabes).
376 Failure of localization. Topoanesthesia	When a cutaneous sensation is felt but cannot be localized.	Allocheiria occurs in hysteria, very rarely in tabes, hemiplegia and sclerosis. Polyesthesia occurs only in tabes and in hysteria.
377 Allocheiria	When an irritation is not felt at the point of contact, but at a corresponding point on the opposite side of body.	Paradoxical sensation has been met with in a number of spinal and cerebral diseases, but is without diagnostic significance.
378 Double sensation and Polyesthesia	When one contact gives rise to two distinct sensations (double sensation) or more (polyesthesia).	Haphalgesia occurs in hysteria. Retardation of conduction of pain occurs only in lesions of peripheral sensory neurons (tabes or multiple neuritis), and is therefore, a very important diagnostic symptom.
379 Paradoxical sensation	The quality of thermic sensation is reversed, a hot body feels cold and vice versa.	Persistence of sensation occurs in lesions of the peripheral sensory neurons (tabes).
380 Haphalgesia	A slight tactile impression from certain objects, but not from others, is felt as intense pain.	Binocular diplopia is due to a weakness of one or more of the external muscles of one eye, or to displacement of one eyeball; so that the image does not fall on identical spots in the two retinae.
381 Retardation of conduction of pain	The sensation of pain is not felt until an appreciable interval after the time of contact.	
382 Persistence of sensation	The sensation continues an unusually long time after the irritation causing it has ceased to act.	

	Diagnostic Symptoms	Definition	Significance
PERVERSIONS (Concluded)	383 Binocular Diplopia (818)	Two separate visual perceptions of the same object, the perception from the normal eye (true image) being more distinct than that from the abnormal eye (apparent image).	Monocular diplopia may occur in hysteria, in cases of double pupillary opening, in anomalous refraction (incipient cataract), and irregularities in the cornea.
	384 Monocular Diplopia or Polyopia (880-4)	A condition in which objects appear double or multiple, even when looked at with one eye alone.	Metamorphopsia may occur in hysteria, also in astigmatism (refractive) and in displacement of the retinal elements (retinal) which may ocur in retinitis, choroiditis, and in detachment, or tumor, of retina.
	385 Metamorphopsia	A condition in which objects appear distorted.	Micropsia may occur in hysteria, in paralysis of accommodation and, with distortion, when the retinal elements are spread apart (recent choroiditis or retinitis).
	386 Micropsia	A condition in which everything looks much smaller than normal.	Macropsia may occur in hysteria, in spasm of accommodation and, with distortion, when the retinal elements are crowded together (atrophic stage of retinitis and choroiditis).
	387 Macropsia	A condition in which everything looks much larger than normal.	Tinnitus aurium, parakusis, parosmia and parageusia occur in lesions of the terminal organ of the uncinate gyrus, and in insanity and functional disorders. They may constitute the aura of an epileptic attack.
	388 Tinnitus Aurium	A sound of ringing, roaring, whistling, etc., in ears or head.	Vertigo may be functional (hysteria, neurasthenia, traumatic neuroses); or may depend on changes in the cerebral circulation, especially anemia and hyperemia (cardiac and arterial diseases, congestion in portal or systemic circulation, galvanism of head or neck), or toxic (tobacco, morphine, alcohol, some digestive disturbances, etc.); or may depend on diseases of the cerebellum and its tracts, or of the ear or eye. It is the principal symptom in Ménière's disease (aural vertigo). Vertigo is closely associated with vomiting. In vertigo associated with lesions in, or pressing upon, a cerebellar hemisphere, external objects seem to whirl in the direction away from the injured hemisphere in both conditions, but the subjective vertigo, usually, is away from the injured hemisphere in case the lesion is within it and towards it when the lesion is external and presses upon the hemisphere.
	389 Parakusis	Perversions of hearing, such as hearing tones incorrectly or hearing better when other loud noises are present at the same time, or hearing sounds or words for which there is no external cause (hallucination).	
	390 Parosmia	The perceptions of abnormal odors or of those for which there is no external cause (hallucination).	
	391 Parageusia	The perception of abnormal tastes or of those for which there is no external cause (hallucination).	
	392 Vertigo	A feeling as if the person (subjective) or as if surrounding objects (objective) were whirling about, or both.	

Diseases in which these conditions occur are discussed in Charts XXIV and XV.

CHART VIIa

Electrical Examination

Comprising Numbers 393 to 403

Analysis of the Symptoms of the Case (Semeiology)

Definition, Significance and Relationship of the Symptoms of Disease

	Name of the Reaction	Tissue Tested	Reaction to Faradism.	Reaction to Galvanism and Formula of Galvanic Reaction	Character of the Contraction	Significance of the Reaction
393 ELECTRICAL REACTION OF MUSCLES AND NERVES (70-3) Nerve fibers respond to changes in intensity of both the faradic and the galvanic currents. The changes in intensity are best brought about by making and breaking the current. Muscle fibers respond only to the galvanic current. The muscle responds to the faradic current only in virtue of the nerve fibers supplied to it. When these nerve fibers are degenerated the muscles can no longer respond to the faradic current. Both nerves and muscles have points on the body surface; the so-called motor points (see figures 1 to 5) from which they are most readily excitable. Therefore, in testing a nerve or muscle by electricity the electrode (positive or negative) is placed on the corresponding motor point. (70-3).	394 Normal excitability (473)	NERVE AND MUSCLE	Contraction present to a strength of current which is normal for the nerve and muscle tested.	Neg.Cl.C. Pos.Cl.C. Pos.Op.C. Neg.Cl.Tet. is the normal formula, or in other words Neg.Cl.C. occurs with the weakest current that will cause any contraction. Pos.Cl.C. occurs with a little stronger current. Pos.Op.C. occurs with a still stronger current. The explanation of the above formula is as follows: The weakest current that will cause any contraction of the muscle will do so when the negative electrode is on the motor point and the current is closed. (Neg.Cl.C.) A more powerful contraction will take place when a stronger current is used and then there will also be a contraction when the current is closed and the positive pole is on the motor point (Pos.Cl.C.). A still more powerful current causes a contraction when the current is opened and the positive electrode is on the motor point (Pos.Op.C.). With such powerful currents and the negative pole on the motor points there results a tetanus or continuous contraction when the current is closed. (Neg.Cl.Tet.); so that the muscle cannot relax to contract again when the current is opened. There is, therefore, in health no reaction corresponding to "Neg.Op.C."	Quick.	Normal excitability shows a normal condition of muscle and nerve.
	395 Diminished excitability		Contraction present but it requires an unusually strong current to produce it.			Diminished excitability occurs in many diseases and conditions (thick skin), especially in lesions of the central motor neurons and is not of much value in diagnosis.
	396 Exaggerated excitability		Contraction present to an unusually weak current.			Exaggerated excitability is a rare condition. It occurs in nervous persons with moist skins and in tetany.
	397 Reaction of degeneration (472)	NERVE	Gradual loss of excitability which becomes complete in about two weeks after injury or onset of the disease.	No reaction.	None.	The reaction of degeneration proves that the peripheral motor neurons are degenerated and that recovery will either never take place, or will be very slow. The lesion must be either in the peripheral nerves or nerve roots or in the anterior horns of the spinal cord or in the motor nuclei in the brain stem.
		MUSCLE	Gradual loss of excitability which becomes complete in less than two weeks after injury or onset of the disease.	After the first two weeks the muscle responds to unusually feeble galvanic currents and the normal formula is reversed; the positive pole being more potent: Pos.Cl.C. Neg.Cl.C. Pos.Op.C. Neg.Op.C. (which last reaction never occurs in health). It is usual to express the formula for the normal reaction and for the reaction of degeneration in the German language in which Kathode means the negative electrode and Anode means the positive electrode. The usual formula is K.C.C., A.C.C., A.O.C., K.C.Te. The reaction of degeneration is A.C.C., K.C.C., A.O.C., K.O.C. The essence of the normal formula is K.C.C. > A.C.C. The essence of the formula of the reaction of degeneration is A.C.C. > K.C.C.	Sluggish. The sluggish character of the muscular contraction is the most characteristic thing in the reaction of degeneration.	

ELECTRICAL REACTIONS (Concluded)

	Name of the Reaction	Tissue Tested	Reaction to Faradism	Reaction to Galvanism	Formula of Galvanic Reaction	Character of the Contraction	Significance of the Reaction
ELECTRICAL REACTION OF MUSCLES & NERVES (Con)	398 Partial reaction of degeneration	Nerve	Contractions present, but require unusually strong currents, whether faradic or galvanic.		Either the normal formula, or the formula of the reaction of degeneration, or a combination of the two may be present. A.C.C. may equal K.C.C.	Quick or Sluggish.	The significance of this reaction is the same as that of the reaction of degeneration, except that it indicates the lesion is less severe and that all the nerve fibers are not degenerated.
		Muscle	Contractions present only to unusually strong currents.	Contractions present to unusually weak currents		Sluggish	
	399 Myasthenic reaction (553)	Nerve and Muscle	Contractions quickly grow less strong and soon cease under rapidly repeated excitation.	Normal	Normal	Quick; grows rapidly weaker and ceases.	Occurs only in myasthenia gravis (554).
	400 Myotonic reaction (613)	Nerve and Muscle	Continuous tonic contraction lasting some time after the electrical stimulation has ceased.	Curious wave-like contractions occur and last after electrical stimulation has ceased.	Positive pole is about equally as potent as the negative. Hence the formula A.C.C. = K.C.C.	Continues usually a *long time* and has a wave-like character.	Occurs in Thomsen's disease (611).
	401 Neurotonic reaction	Nerve	Unusually excitable. Tetanic contraction persists after electrical stimulation has ceased.		Normal.	Continuous	Occurs in hysteria, amyotrophic lateral sclerosis and chronic bulbar paralysis.
		Muscle	Normal.	Normal.			
	402 Reaction of completely degenerated muscle (70 to 73)	Muscle	None.	None.	None.	None.	Muscle fibers are entirely degenerated and recovery is impossible.

403 Electrical reaction of the Optic and Auditory Nerves

- The optic nerve responds to the galvanic current with a sensation of light, the color of which varies with the pole employed.
- The auditory nerve responds with a loud sound when the negative electrode is placed in or near the meatus and the current closed and with a faint sound when the positive pole is used and a stronger current broken. These reactions are without diagnostic importance.
- The negative electrode placed in front of the ear causes a nystagmus towards the ear tested when the current is closed and in the opposite direction when the current is broken. The positive electrode causes nystagmus in exactly the reverse direction.

In cases of disease in which the caloric test (79) is absent and in which the electric test is present, it is fairly certain that the lesion is in the labyrinth and not in the nerve. If there is no response to either the caloric or the electric test the lesion is in the nerve or its nucleus.

CHART VIIb

Erb's Motor Points for Electrical Examination of Nerves and Muscles

The motor points are the areas upon the surface of the body at which the individual nerves and muscles can be most easily excited by electricity. For the nerves, these points coincide with those at which the nerve lies most superficially or where it can be pressed against a resisting tissue; for the muscles, they lie over the point of entrance of the nerve into the muscle.

Comprising Figures 1 to 5

ERB'S MOTOR POINTS

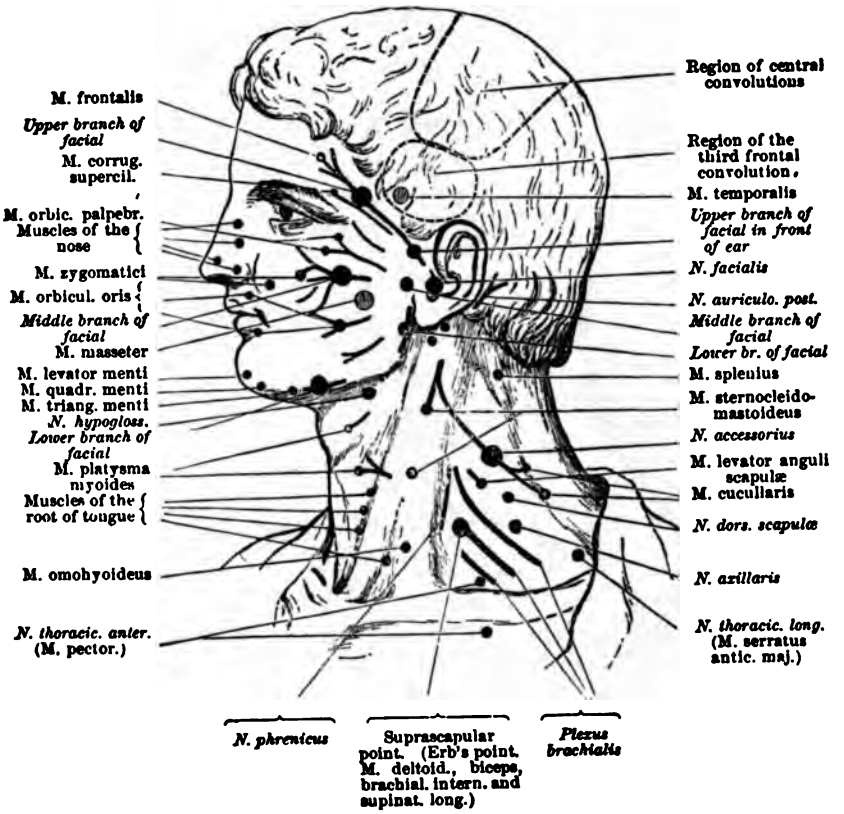


FIG. 1

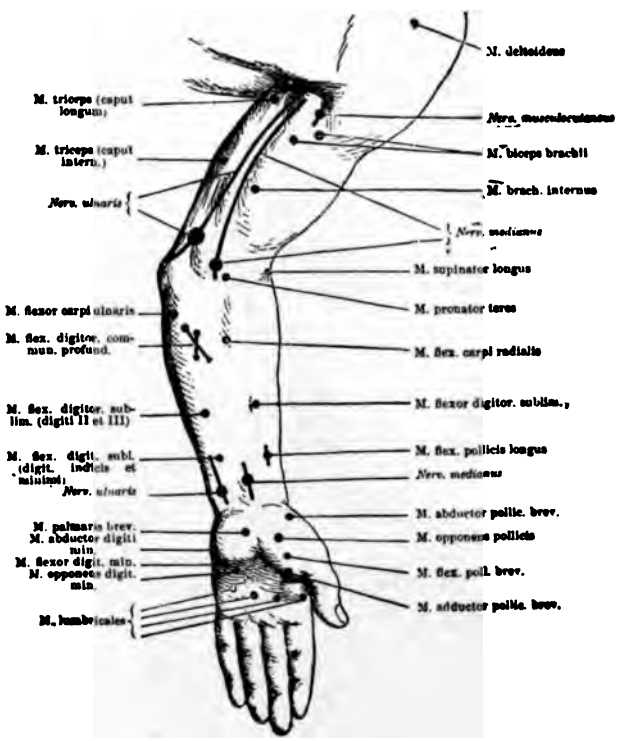


FIG. 2

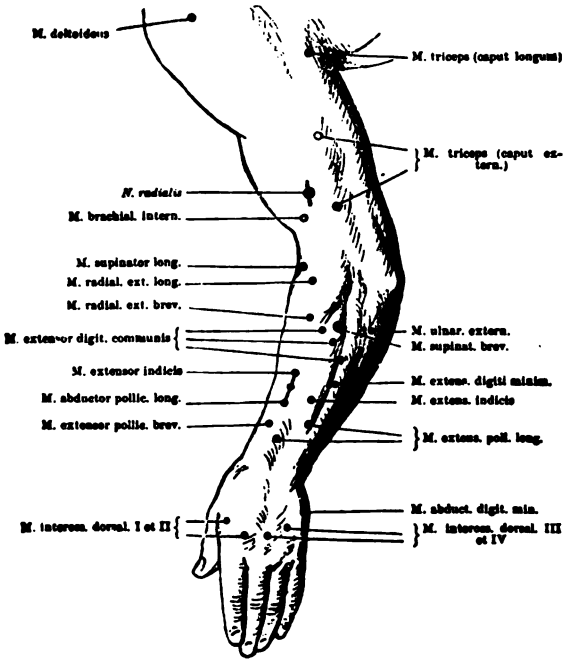
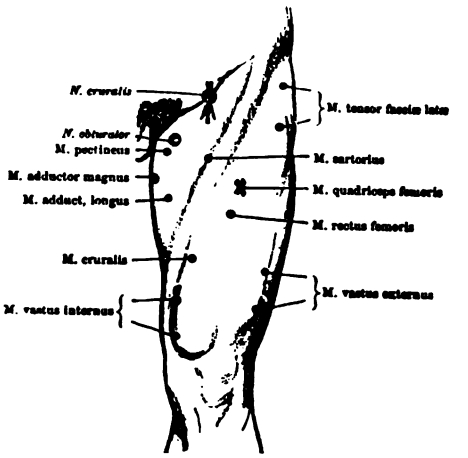


FIG. 3



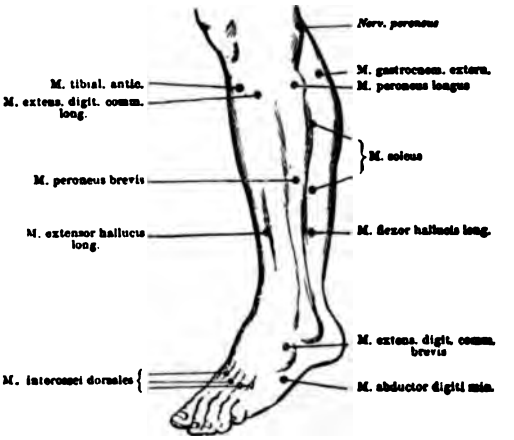


FIG. 4

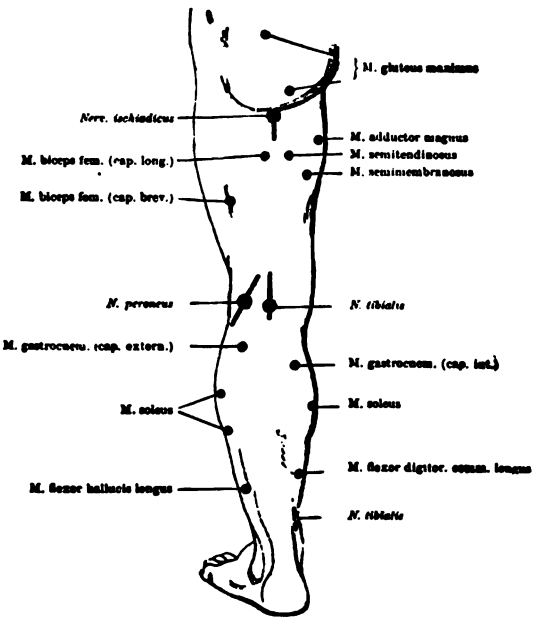


FIG. 5

CHART VIIc

ERB'S DIAGRAM SHOWING THE EFFECTS OF INJURY OF A NERVE

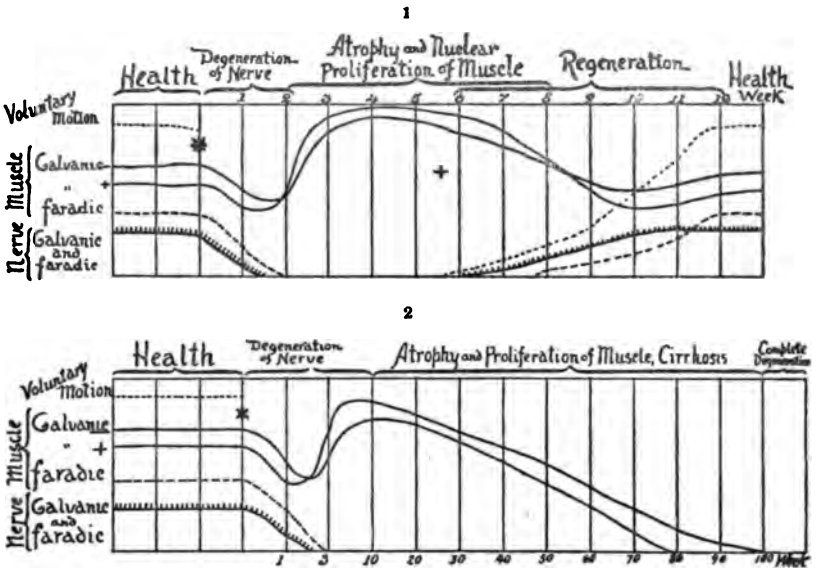


FIG. 6

Charts Illustrating the Reaction of Degeneration

The star (*) indicates the incidence of a paralysing lesion in the domain of the peripheral neuron. Voluntary motion is lost at once. During the first two weeks there is slight diminution of the galvanic excitability of muscle; there is also rapid diminution of the faradic excitability of muscle and of the galvanic and faradic excitability of nerve, which are completely lost at the end of the second or third week. During the second week there is rapid increase in galvanic excitability of muscle and the response to the positive pole becomes greater than to the negative.

Chart 1 represents the reaction in a case terminating in recovery. During the sixth week (indicated by the cross X) regeneration begins. The increased galvanic excitability of the muscles gradually diminishes until it becomes normal and the poles are reversed so that the negative response is again greater than the positive. Voluntary motion returns first, then the galvanic and faradic excitability of the nerve, and last of all, the faradic excitability of the muscles.

Chart 2 represents the reaction in a case terminating in atrophy and cirrhosis of the muscle. The galvanic excitability of the muscle is increased and the poles are reversed, as before. The decline in galvanic excitability continues, however, until the end of the second year, when it is entirely lost. Voluntary motion, and the electrical reactions of both muscles and nerve are permanently destroyed.

CHART VIII

Analysis of the Cerebro-Spinal Fluid

Comprising Numbers 405 to 414

ABNORMAL CEREBRO-SPINAL FLUID

404 ABNORMAL CEREBRO-SPINAL FLUID

Characteristics	Methods of Testing	Significance
405 Tension	Can be roughly estimated by the rapidity of flow of fluid through the canula, whether in drops or a stream; more accurately by the height to which the fluid rises in a vertically held glass tube connected by a short rubber tube with the canula. A stopcock on the canula adds to the accuracy by preventing the escape of much fluid and the consequent lowering of the tension. The fluid in the tube rises and falls with the respiration. An additional more rapid and stronger pulsation indicates a basilar aneurism.	A low or very rapidly diminishing tension has no diagnostic meaning, except as indicating an obstruction to the communication of the fluid in the ventricles with that of the vertebral canal, as in closure of the foramen of Magendie. A high tension means increased intra-cranial, or intra-spinal, pressure caused by an increased amount of cerebro-spinal fluid or by a foreign body within the cerebro-spinal cavity. It occurs in tumors, abscess, hydrocephalus, hemorrhage, acute, sub-acute and some cases of chronic and serous meningitis, also in cerebral edema (nephritis, anemia, etc.), acute infectious diseases and some other conditions.
406 Red or reddish yellow color	By sight. Hematoidin crystals may be seen under the microscope.	Fresh blood in the fluid may be the result of puncture of a blood vessel, in which case it is most abundant in the fluid first drawn, usually coagulates, and settles quickly on centrifugalization. Or May be the result of hemorrhage into the ventricles or membranes (Hematorhachis, hematoma, aneurism, etc).
407 Cloudy	By sight. Pus cells under the microscope. (Polymorphonuclear leucocytes)	An increase of cellular elements in the fluid is usually the result of an acute or sub-acute meningitis. In some cases of acute meningitis, however, the fluid may be clear.
408 Clear with delicate coagulum	By sight.	Tuberculous meningitis, usually.
409 Cellular elements and bacteria	Fluid soon after withdrawal should be centrifugalized. Tube should be emptied quickly and from its walls and bottom sediment should be sucked in and out of a capillary tube, well mixed and spread on two clean slides. One slide should be stained by Gram's method for bacteria, especially a smear of the fine pellicle found in some clear fluids, in which tubercle bacillii are found in 90% of the cases and the other by Wright's blood stain for cellular elements. Or The fluid (not centrifugalized), 10 parts, can be mixed with 1 part of a solution consisting of methylene blue 0.2%, glacial acetic acid 4.0%, and water to 100%, and counted in a Thoma-Zeiss chamber. Or Rinse out the white counting pipette with glacial acetic acid, draw in well-shaken freshly drawn cerebro-spinal fluid and count.	The normal cerebro-spinal fluid shows under these conditions 1 to 5 cells in a field of the microscope. If there are more than 6 to 8 cells in a field it indicates a meningitis. If the cells are mainly polymorphonuclear leucocytes it indicates epidemic cerebro-spinal, or purulent, meningitis, or rarely an acute tuberculous meningitis: broadly speaking, an acute infectious meningitis. If the cells are mainly or entirely lymphocytes it indicates a tuberculous meningitis, or cerebro-spinal syphilis, or paresis, or tabes, or acute anterior poliomyelitis, encephalitis or convalescence from any form of acute meningitis: broadly speaking a chronic infectious meningitis. If echinococcus cysts or hooklets are present, they indicate the presence of an echinococcus cyst.

ABNORMAL CEREBRO-SPINAL FLUID (Concluded)

Characteristics	Method of Testing	Significance
410 Sugar	By Haines' test or other tests.	Not of much significance, but the sugar normally present is diminished, usually, in meningitis and in some other conditions.
411 Albumen	Two c. c. of the fluid mixed with 10 c. c. of Esbach's fluid is centrifugalized during one hour in a conical tube graduated to 0.1%.	Normally not more than ½% is present. Usually increased in meningitis and tumors. A diminution in the amount usually indicates a progressive space-occupying disease. Of little diagnostic significance.
412 Globulin	Two c.c. of a saturated solution of chemically pure neutral ammonium sulphate should be placed in a test tube and one c. c. of the cerebro-spinal fiuid should be gently run upon its top. If the reaction is positive, within 3 minutes, a grayish white ring should form at the junction of the two fluids. At the end of one-half hour, the surface of the ring should show a delicate network. Best seen by indirect illumination. (Nonne-Apelt test.) Or Boil slightly 1 volume of the cerebro-spinal fluid with 5 volumes of a 10% butyric acid solution, add 1 volume of a normal solution of sodium hydroxide, reheat and allow to cool. If a flocculent percipitate forms, the reaction is positive. (Noguchi test.) The globulin test is of little or no value, if the fluid contains red blood.	Indicates meningitis, acute anterior poliomyelitis, encephalitis, cerebro-spinal syphilis, paresis, tabes, rarely a brain tumor.
413 Positive Wassermann reaction	This test can only be performed in a laboratory by an expert.	The reaction is positive in 90% of cases of paresis and in 60% of cases of tabes. In cerebro-spinal syphilis both the cerebro-spinal fluid and the blood usually give a positive reaction. In other cases of syphilis (without meningitis) the reaction is usually negative with the cerebro-spinal fluid, but positive with the blood.
414 Colloidal gold test (Lange)	This test can only be performed in a laboratory by an expert.	This test is to be regarded, and used, only as an additional or confirmatory test. It is of much value in syphilitic diseases of the central nervous system, especially tabes and paresis. It is positive in about 80% of the proved cases.

(Note)—In the examination for cellular elements (409), it is important to always check up the type of the cells present and the presence of blood cells by centrifuging the cerebro-spinal fluid and staining a drop of the sediment by Wright's stain. It is especially important to thus differentiate resistant red blood corpuscles from small lymphocytes; so that the former may not be counted with the latter, and in interpreting the results of the globulin and colloidal gold tests; both of which tests are vitiated by the presence of blood. This method gives also a surprisingly accurate estimation of the number, as well as of the type, of cells present.

CHART IX

Special Syndromes and Anatomical Terms

Comprising Numbers 415 to 465

Syndrome	Definition	Significance
415 Hysterical symptoms (1076)	Occur usually in self-conscious females of an emotional nature. Lack of inhibition and great susceptibility to suggestion. Desire to excite admiration and sympathy and wonder. Mental instability. Globus hystericus (416). Spinal, inguinal, (or ovarian) and other tenderness. Great variety of symptoms (especially subjective) which cannot be explained by any organic lesion. Glove and stocking form of anesthesia or hemianesthesia and concentric contraction of the field of vision are common symptoms, but the patient is usually ignorant of their existence until they are discovered, or more probably suggested, by the physician. This glove and stocking form of anesthesia rarely occurs also in multiple neuritis and syringomyelia. Exaggerated reflexes but no ankle-clonus or only pseudo-clonus. Never a Babinski reflex. Motor paralyses, tremors, contractions and convulsions are not uncommon. Transference of hemianesthesia can be affected in some cases by suggestion or by the application to the anesthetic areas of metal discs, especially those made of gold. The anesthesia of the fingers does not prevent delicate acts being done by them with eyes closed. Such patients when tested and the anesthetic area is touched often answer "no" at the moment of contact (Janet's test, 48). Many other symptoms do not seem to be real but rather seem to be imaginary and may result from hallucinations or delusions or more probably are the result of suggestion: auto- or foreign. Probably many of the hysterical symptoms come into existence as the result of the physician's careful and minute examination or repeated examinations (foreign suggestion). Many other symptoms in addition to those above mentioned are met with in hysteria. Among the more important are: blindness, coma, aphonia, astasia and abasia, anorexia, vomiting and regurgitation of food, tympanites, phantom tumors and false pregnancies, hemoptysis, anuria and melanuria. Many of these symptoms are pure "fakes." They even drink their own urine, so great is their desire to excite wonder.	Hysteria (1076)
416 Globus Hystericus (1076)	The feeling of a lump or ball behind the upper end of the sternum which interferes both with swallowing and breathing. The sensation often commences in the epigastrium and rises to the base of the neck and remains there; the patient not being able to get it up or down. It may be caused by a spasmodic contraction of the muscles of oesophagus or throat.	
417 Hysterogenic areas (1076)	Spots scattered over the body, but usually in the left inguinal region, where light pressure or irritation will cause more or less violent hysterical attacks.	
418 Hysterofrenic areas (1076)	Spots scattered over the body, but usually in the left inguinal region, where firm and continued pressure will cause the arrest of an existing hysterical attack.	
419 Lasègue's symptom (1076)	A condition in which the patient cannot move an anesthetic extremity when her eyes are closed, but can move it readily when she opens her eyes and looks at it.	
420 The epileptic aura (1061. 575, 849)	The aura is a symptom (warning) which occurs before the attack in about half the cases of epilepsy. It may be remote or immediate. The former is often called "a prodromal symptom" and occurs hours and days before the attack. It consists usually in an emotional change (irascibility, etc.), changes in the amount of sleep, of food taken, in sexual desire and vasomotor phenomena. Much more characteristic and important is the immediate aura which occurs a fraction of a minute before the attack. This aura may be "psychic" (anxiety, anger, joy, dreamy states, special thought or memory, etc.), or a "sensory hallucination"	Epilepsy (575, 849, 1061)

Syndrome	Definition	Significance
	which may be visual (blindness, lights, colors (red), elaborate false visual perceptions, etc.) or auditory (deafness, noises, and false auditory perceptions) or olfactory or gustatory hallucinations or cutaneous paresthesiae (the feeling of a wind blowing on some part of the body is quite common) and pains or visceral paresthesiae, especially epigastric. Vertigo is a common immediate aura; or the immediate aura may be motor and consist in twitching of a group of muscles, (Jacksonian epilepsy), or in more complicated automatic movements of the body, or in hiccough, sneezing, yawning or swallowing. Vasomotor disturbances, flushing or pallor with secondary paresthesiae, are not uncommon immediate aurae. Usually the aura is always the same in the same individual; rarely it varies. In rare cases the aura may not be followed by an attack and in still rarer and always doubtful cases it may be the only symptom of epilepsy.	
411 Jacksonian epilepsy (587-8, 602)	A clonic spasm of one or more muscles in one side of the face or in one arm or leg, which may remain local, but usually rather rapidly extends to other muscles of the same side of face, or of the arm or leg in which it commenced. It then may extend to an adjacent extremity in the same order in which the cortical centers are placed: thus from the face to the arm and then to the leg, from the leg to the arm and then to the face, from the arm to the leg and face nearly or quite simultaneously, but never from the face to the leg, or vice versa, without involving the arm. When the spasm has extended over the whole half of the body it may remain so or may pass across and involve the other side. As long as the spasm is local or limited to one-half of the body consciousness may or may not be lost, but when the spasm involves both sides of the body consciousness is always lost.	Local cortical lesion (587-8, 602) (Figs. 15-16)
422 The prodromata of apoplexy (504, 1063-6)	In many cases of apoplexy, especially in cases of cerebral thrombosis, the apoplectic attack is preceded by a number of more or less definite and characteristic symptoms which may be remote, preceding the attack by months or years; or immediate, occurring immediately before the attack. These prodromata are both *general*, such as headache, vertigo, drowsiness and stupor, irritability, forgetfulness, hypochondriacal feelings, ringing in the ears, flashes before the eyes. etc.; and *local*, such as temporary attacks of aphasia, diplopia, achromatopsia, dysarthria, temporary paralysis of arm or paresthesiae. None of these symptoms is so characteristic that an attack of apoplexy can be confidently predicted from its presence. The most constant prodromal symptom of apoplexy (except of embolism) is high arterial tension.	Apoplexy (504, 1063-6)
423 Tabetic or visceral crises (661)	Paroxysmal attacks of pain in, and functional disturbances of, some viscera, occurring in the course of locomotor ataxia. These attacks recur after irregular intervals, persist during an hour, or a day or two, and are analogous to the paroxysmally occurring lightning-like pains in the legs. "Gastric crises" are the most frequent and consist in severe pain in the epigastrium together with uncontrollable vomiting and retching. At times attacks of gastric pain or of vomiting occur separately. "Hepatic crises" resemble gallstone colic, even being accompanied by slight jaundice at times. "Laryngeal crises" consist in attacks of coughing and dyspnoea. "Laryngeal vertigo" (Ictus laryngeus) consists in a sensation of tickling and burning in the larynx, a stridulous inspiration with a feeling of suffocation and a falling to the ground unconscious for a few minutes. "Pharyngeal crises" consist in repeated acts of noisy swallowing. "Renal crises" resemble attacks of renal colic. "Vesical crises" consist in pain in region of bladder and prostate, and constant desire to urinate. "Urethral crises" consist in attacks of pain in urethra and desire to urinate. "Rectal crises" consist in attacks of pain in the rectum and tenesmus. "Vulva-vaginal crises" consist in attacks of pain in vagina. "Clitoridean crises" consist of attacks of pain in vulva with sexual desire and discharge of mucus. "Anginal crises" resemble angina pectoris. Occasionally "crises" of several kinds occur simultaneously.	Tabes (661) (Fig. 27)

Syndrome	Definition	Significance
424 Bulbar symptoms (546)	A combination of several or all of the following symptoms: dysarthria or anarthria (283-4), dysphagia (285), drooling of saliva from mouth, propulsive speech, and puffing of lips. Paralysis of the 7th, 9th, 10th, 11th, and 12th, and at times of other cranial nerves. Spastic paraplegia or hemiplegia of extremities. Sensory paralyses and ataxia. Respiratory difficulty, and in severe cases rapid, irregular pulse and Cheyne-Stokes' respiration.	Lesion or disorder of medulla (546). (Figs. 21-2)
425 Cheyne-Stokes' respiration (728)	Long pauses in the respiration. After a pause the respiration commences slow and deep and rapidly becomes quick and superficial and as rapidly becomes slow and deep again and terminates in another long pause (lasting from five to sixty seconds, or more) and so on; each cycle being completed in a few minutes. A somewhat similar respiratory disturbance which is called Biot's respiration consists of frequent pauses in the respiratory act, lasting many seconds. Biot's respiration occurs in Bright's disease, etc., but has no particular significance in nervous diagnosis.	
426 Stokes-Adams' phenomenon (582, 1060)	Slow pulse with long arrests (one-half to one minute or more) during which the patient becomes pale, unconscious and may show a more or less pronounced convulsion.	Lesion of bundle of His in the heart, or irritation of the pneumogastric nerve.
427 Babinski and Nageotte's bulbar syndrome (1268)	Paralysis of the tongue, diaphragm and larynx with ataxia of the homolateral side; analgesia and thermic anesthesia with motor paralysis of arm and leg of the contralateral side, myosis and pseudo-ptosis, dysphagia and dysarthria.	Lesion of medulla. (Figs. 21-3)
428 Ponto-cerebellar angle syndrome (1377)	Homolateral deafness and contralateral analgesia and thermic anesthesia with preservation of tactile sensibility, nystagmus, weakness of conjugate deviation of the eyes towards the side of the lesion, anesthesia and abolition of reflexes in the distribution of the trigeminus on side of lesion, adiadocokinesia on the same side, optic neuritis, cerebellar ataxia and occipital pains, all more marked on side of lesion.	Lesion at ponto-cerebellar angle. (Fig. 20)
429 Millard-Gubler's syndrome (1292)	Homolateral facial paralysis with contralateral paralysis of arm and leg.	Lesion of pons. (Fig. 20)
430 Weber's syndrome (1293)	Homolateral oculo-motor paralysis with contralateral hemiplegia.	Lesion of crus cerebri.
431 Benedykt's syndrome (1293, 1341)	Homolateral oculo-motor paralysis associated with a tremor of the contralateral arm and leg.	Lesion of red nucleus or of rubro-spinal tract.
432 Brown-Séquard's paralysis or spinal hemiplegia (509, 844, 982)	Below the point of lesion there are motor paralysis, exaggerated tendon reflexes, Babinski reflex, elevation of temperature, vaso-motor disturbances, and at times more or less hyperalgesia, ataxia, and loss of deep sensibility on the homolateral side, together with analgesia, thermic anesthesia, apallesthesia (353) and more or less tactile anesthesia, on the contralateral side. The anesthesia is bounded above by a narrow zone of hyperesthesia or hyperalgesia. Brown-Séquard's paralysis is more often atypical than typical.	Unilateral spinal lesion. (Figs. 24-6)
433 Spinal epilepsy (60-1 and 520)	Violent and continued tremor of the leg after it has been struck or shaken.	Greatly exaggerated tendon reflexes.
434 Bell's phenomenon	A turning upward of the eyeballs when an attempt is made to close the eyelids in peripheral facial paralysis.	Facial paralysis (peripheral).

Syndrome	Definition	Significance
435 Strümpell's tibialis phenomenon	When a patient, with spastic paralysis of a leg, lying on his back, attempts to flex the paralysed leg at the knee against light resistance, a dorsal flexion of the foot also occurs. Strümpell has found similar phenomena in the radial and pronator groups of muscles in the forearm.	Lesion of the pyramidal tract. (Figs. 24-6)
436 Babinski's associated movements of trunk and thigh	When a patient with spastic paralysis of one leg, lying on a hard surface without a pillow, with legs slightly abducted and hands folded across chest, attempts to raise the body to a sitting posture, the paralysed leg is involuntarily raised from its support while the normal leg lies at rest. This movement does not occur in hysterical paralysis.	
437 Argyll-Robertson pupillary reflex (891)	Loss of the pupillary reflex to light, while the reflex persists with efforts of accommodation and the consequent convergence and parallelism of eyeball (332.)	Tabes, paresis and syphilis (661).
438 Romberg's symptom (static ataxia)	A wavering, staggering and even falling when attempting to stand still with eyes shut and with the feet in contact, either laterally or the one before the other (42.)	Cerebellar disease (647).
439 Biernacki's sign	A loss of the normal sensitiveness to pressure of the ulnar nerve behind the elbow.	Tetany (614).
440 Trousseau's sign	Pressure on the nerve trunks of the extremities causes a tetanic spasm of the muscles supplied by them.	
441 Chvostek's sign	The facial nerve shows extreme irritability to percussion or pressure.	
442 Erb's sign	Muscles and nerves are unusually excitable both to galvanism and to faradism.	
443 Quinquand's sign	Patient spreads his fingers and presses their tips against the palm of the observer's hand which is held vertically. After a few seconds a series of slight shocks are felt as if the phalanges of each finger were knocking together.	Chronic alcoholism.
444 Erb's paralysis. Combined shoulder and arm paralysis (490)	A paralysis of the deltoid, biceps, brachialis anticus and supinators, long and short. In some cases the supra-and infraspinatus muscles are also paralyzed, and to a less extent the extensors of the wrist and fingers. Anesthesia of outer aspect of forearm and hand is occasionally present. Paralysis of 5th and 6th cervical nerve roots.	Lesion of the brachial plexus. Erb's paralysis may be due to injury at birth (obstetric paralysis.)
445 Klumpke's paralysis (490)	A paralysis of the small muscles of the hand and fingers. There is anesthesia of ulnar side of forearm and hand. In some cases the muscles of the forearm, except the supinator longus, are also paralyzed, and the eye on the same side exhibits miosis, retraction of the bulb and narrowing of the eyelid opening. Paralysis of nerves arising from the 7th and 8th cervical and 1st dorsal nerve roots.	
446 Brudzinski's neck sign	When the arms and legs are flexed fully on the trunk and the head is passively bent forward the patient shows signs of pain.	Meningitis.
474 Brudzinski's leg sign	When one leg is passively fully flexed on the trunk the other leg is drawn up by the patient into a similar position.	

Syndrome	Definition	Significance
448 Grasset and Graussel's phenomenon	Inability of a patient when lying on his back to raise both legs simultaneously although he is able to raise either leg separately.	Organic hemiplegia (incomplete)
449 Avellis' syndrome	Recurrens paralysis of one side with paralysis of the uvula of the same side.	Lesion of nuclei in the medulla (706)
450 Schmidt's syndrome	Same as Avellis, and also paralysis of the trapezius and sterno-cleido-mastoid muscles of the same side.	
451 Korsakoff's psychosis	Retroactive amnesia (769). Failure to appreciate relations of time and space.	Alcoholism (1102)
452 Bristowe's syndrome	Progressive hemiplegia with vague hemiplegic symptoms on the other side. Drowsiness increasing to coma, dysphagia and dysarthria, but no other implication of the cranial nerves.	Lesion of the corpus callosum (1300)
453 Brun's syndrome	Vertigo associated with movements and change of position of head.	Intra-ventricular lesion (1008)
454 Foville's syndrome	Paralysis of face and of oculo-rotary power towards the same side and of arm and leg of the opposite side.	Lesion of tegmentum of pons (1292)
455 Horner's syndrome	Miosis, ptosis, enophthalmos and anhydrosis.	Paralysis of cervical sympathetic ganglion (1191)
456 Schüller's side-gait	The patient when stepping laterally along a straight line walks badly in both directions.	Hysterical hemiplegia (796)
	The patient when stepping laterally along a straight line walks toward the paralysed side and badly towards the healthy side.	Organic hemiplegia (797)

ANATOMICAL TERMS

460 Brain stem	Comprises the medulla oblongata, pons Varolii and crura cerebri. (Figs. 18-23.)
461 Cortico-spinal or upper motor neurons	Motor cerebral cortex, corona radiata, internal capsule, pyramidal tracts at base of brain, motor decussation and crossed and direct pyramidal tracts in spinal cord. (Figs. 15-26.)
462 Spino-muscular or lower motor neurons	Motorial end plates, peripheral nerves, anterior nerve roots, nerve cells in the anterior horns of spinal cord and the motor nuclei in the brain stem. (Figs. 19, 26.)
463 Central sensory or upper sensory neurons	Sensory cerebral cortex, corona radiata, internal capsule, cerebellum and its peduncles, lemniscus and sensory decussation, nuclei of columns of Goll and Burdach, antero-lateral ascending (Gower's) tract, direct cerebellar (Flechsig's) tract and column of Clark. (Figs. 15-26.)
464 Peripheral sensory or lower sensory neurons.	Sensory end organs, peripheral nerves, posterior nerve roots, spinal ganglia, posterior horns and columns of Goll and Burdach in the spinal cord and nuclei of columns of Goll and Burdach. (Figs. 22-6.)
465 Cilio-spinal center (335, 1191-2)	Situated in the lateral horn of gray matter in the last cervical and first dorsal segment of the spinal cord and is connected with a higher center in the medulla. Destructive lesions of this center and its nerve roots cause (1st) a paralytic miosis, (2d) a narrowing of the eyelid opening, (3d) an enophthalmos; while irritative lesions (rare) of this center and its nerve roots cause (1st) a spasmodic mydriasis, (2d) an exophthalmos (Homer's Sydrome—455).

PART II

Differential Diagnosis

A Clinical Diagnostic Analysis of the Symptoms

OBTAINED FROM THE EXAMINATION OF PATIENTS

Introduction to the Diagnostic Charts

DIRECTIONS FOR THEIR USE.

In using this book for diagnostic purposes the student, or practitioner, having made a complete examination of the patient according to the scheme presented in chart I, should make note of the more important symptoms. Then, selecting any one of these symptoms, he should turn to the table of contents and see which chart treats of the disorders which include this symptom. Finally, turning to the *commencement* of the chart indicated, he should apply one test after another until he reaches the diagnosis.

At the left margin of each chart is placed the symptom to be analyzed; on the right margin are placed all the possible diseases in which this symptom can occur. Proceeding from left to right, in each column a number of alternatives are offered, and by selecting the one appropriate to the case the diagnostician proceeds from one column to the next, until he reaches the correct diagnosis. In the column immediately preceding the diagnosis is an abstract of most of the symptoms which may occur at different stages of the disease. The great majority, but not necessarily all, of the symptoms given in the abstract should be present in the history or found in the examination of the case, if the diagnosis be correct.

A few practical examples will illustrate the method much better than a long general description. Let us, then, consider a few cases as they occur in actual practice. Only the essential symptoms are noted.

Case I. Male, aet. 51.—He smoked and chewed tobacco and drank to excess for years. About two months ago he began to have pains at various points in both legs. His legs became slowly weaker and his flesh became tender, but he is able to walk a mile. Organic reflexes normal. Feet cold, and the legs have lately grown smaller. On physical examination the muscles of the lower legs, and less so those of the thighs are weak, tender and somewhat atrophied. The legs, and especially the feet show slight anesthesia, marked analgesia and well marked retardation of the conduction of pain. Achilles reflex absent. A slight knee-jerk can be obtained with difficulty. In walking toes drop a little and the knees are raised abnormally high.

Important symptoms: PARALYSIS (weakness), HIGH-STEPPING GAIT, ANESTHESIA and PAIN.

The chief symptom in this case is weakness, and we, therefore, turn to chart X, which discusses "diseases causing motor paralysis." The paresis in this case is continuous and the reflex acts are diminished or absent. We, therefore, have to do with a flaccid paralysis and turn to chart Xa. The presence of muscular atrophy following the paralysis, together with the absence of any apparent hypertrophy, guides us in the second column away from the functional diseases and the muscular dystrophies and to the degenerative atrophies; while the normal organic reflexes guide us in the third column away from the spinal cord, and to the peripheral nerve, diseases. The presence of anesthesia, pains, muscle tenderness and other sensory symptoms guides us in the fourth column to the class of neuritis of the spinal nerves. In the fifth column the fact that there are many spinal nerves affected guides us to the diagnosis: Multiple Neuritis, which the history of alcoholic abuse confirms.

We can approach this case in another way by considering his abnormal walk. In the table of contents we find that disorders of gait are treated in chart XIII and indeed in chart XIIIc. The walk in this case is evidently "paralytic and flaccid," the tendon reflexes being diminished. Furthermore it is a high-stepping gait. A comparison of the three possible abstracts with the symptoms of our case makes it evident that the case is one of Multiple Neuritis.

We can trace the case also by means of the anesthesia and analgesia: symptoms which are discussed in chart XIVa. The tendon reflexes being diminished and the organic reflexes normal in this case, we are led to three abstracts, only one of which fits our case, and thus the diagnosis of Multiple Neuritis is again confirmed. Finally we may take up the initial symptom in the case: pain in the legs. Pain is discussed in chart XV and pain in the extremities in chart XVc. In our case the pain is bilateral and is associated with anesthesia; so that we are again brought to three abstracts, of which the one of Multiple Neuritis most nearly fits our case.

Case II. Female, aet. 23.—Ten years ago she and her brother had simultaneously an attack of headache, backache and fever. Her brother died and she recovered with a paralysis of both legs, which has since improved, rapidly at first, then more slowly. Her legs are still somewhat weak, especially the left one, but she can walk fairly well. No sensory disturbances, organic reflexes normal. On physical examination there is a decided weakness, slight atrophy and slight shortening of left leg. Knee-jerks are absent in both legs. No objective sensory abnormalities.

Important symptom: PARALYSIS.

In the analysis of this case we follow the same path traced in case I until we reach column four in chart Xa. In this case there are no sensory symptoms, the paralysis involves neither the cranial nor the extensor nerves exclusively, and is acute in its origin; so that the diagnosis must be Acute Anterior Poliomyelitis.

Case III. Male, aet. 48.—Had a chancre followed by a cutaneous rash twenty-two years ago. During the past ten years has had "lightning pains" in legs and a girdle sensation, also gastric, vesical and urethral crises. During the past six months, his walking has become difficult and awkward and is much worse, practically impossible, in the dark. Organic reflexes normal, except for some delay in micturition. On physical examination there is no loss of muscular power, but all movements of legs are awkward, violent and excessive. There are marked ataxia, anesthesia in areas and well marked retardation of conduction of pain from feet. Complete absence of knee-jerk. Argyll-Robertson pupillary reflex, Romberg's symptom and loss of muscle sense in legs. Lumbar puncture gave fluid showing the pressure of globulin and lymphocytosis and a positive Wassermann. In walking the patient does not stagger, but flings feet out widely.

Important symptoms: ATAXIA, ATAXIC GAIT, ANESTHESIA, PAIN, ABDOMINAL CRISES, and LYMPHOCYTOSIS IN CEREBRO-SPINAL FLUID.

In this case there is no loss of motor-power but well marked ataxia in legs. From the table of contents we learn that diseases causing perversion of motion, including ataxia, are treated in chart XIIa, to which we turn. As the patient does not stagger in walking and the movements of the legs are ataxic, not only in walking, but also in other movements, it is certain that the case is one of "motor ataxia." The ataxia is bilateral and the knee-jerks are absent; so that it is evident that we have to do with tabes or multiple neuritis (pseudo-tabes). We differentiate these two diseases by comparing the abstracts of their symptoms. As in this case there are no muscular weakness, atrophy and tenderness, it is plain that the diagnosis is Tabes.

We may also reach a diagnosis in this case by studying the patient's walk with the aid of chart XIIIc. The gait is ataxic, rather incoördinated than staggering, the knee-jerks are abolished and there is Argyll-Robertson phenomenon; so that the diagnosis of Tabes is confirmed. Furthermore we may trace the case by the symptom of anesthesia with the aid of chart XIVa. The tendon reflexes are abolished. The organic reflexes are not much disordered, but they are slightly. There is no motor paralysis and thus we are led again to Tabes. If we consider the symptom named "Argyll-Robertson phenomenon," which is present in this case, we shall find it discussed in chart XIVd and here again we are led directly to Tabes. If we consider the pains in the legs or the girdle sensations about the body or the abdominal crises, we find them discussed in chart XV and in either case are led to Tabes. If we consider the results of the examination of the cerebro-spinal fluid with the aid of chart XIX, we find the butyric acid test positive, the existence of lymphocytosis, a positive Wassermann, a clear fluid and ataxia, and thus the diagnosis of Tabes is again confirmed.

Case IV. Female, aet 19.—Patient's father and mother were first cousins. They had eight children, of whom three died in infancy and four are healthy. Child learned to walk late and with difficulty, frequently stumbled and fell. Was backward at school and when she was nine years old it was evident to all that she was not normal. Patient's movements became gradually and steadily more awkward. Now she cannot walk without aid. General movements are slightly ataxic and simulate somewhat tremor. Movements of the legs are more ataxic and weaker than those of the arms. Her walk is extremely ataxic and staggering. No knee-jerks, Babinski present. Organic reflexes normal. Internal strabismus. No loss of muscle sense.

Important symptoms: ATAXIA and ATAXIC GAIT.

The most characteristic symptom in this case is ataxia and so, as in case III, we turn to chart XIIa. In this case, the ataxia is mainly on walking and there are no motor paralysis and no loss of muscle sense. We are, therefore, brought to the alternative as to whether the disease occurs in an adult or a child. This case doubtless dates from early childhood. There are no similar cases among her brothers and sisters, but she comes certainly from a tainted family. She has no nystagmus, but has strabismus. As this case began before puberty and has no knee-jerks it is doubtless a case of Friedreich's Ataxia. The strabismus points to Marie's hereditary cerebellar disease and indeed these two diseases are so closely related that there is some question as to whether they are separate entities.

We may approach this case from a different angle. The chief symptom is difficulty in walking. We turn, therefore, to chart XIIc and note that the walking is ataxic. The staggering gait which is permanent, the bad heredity, the absence of knee-jerk and the commencement of the disease in infancy confirms the diagnosis of Friedreich's Ataxia. It may be noted in passing that this case does not show a distinct tremor, or nystagmus, or the blurred speech which symptoms are often present in this disease.

Case V. Male, aet. 62.—His disease commenced with difficulty in speaking and swallowing about a year ago, and has slowly and steadily progressed. His speech has become so bad that it is unintelligible and he has the greatest difficulty in swallowing, and chokes over his food. There is constant drooling of saliva from his mouth. Cannot protrude his tongue beyond his teeth, cannot raise his arms because of weakness of muscles about the shoulders. His legs are somewhat weak. Fibrillary contractions and great atrophy of muscles of tongue and of shoulder girdle (deltoid, pectorals, etc.). Muscles of hands are not involved. Absence of tendon reflexes in arms. Knee-jerks lively, ankle-clonus and Babinski are present. There are no sensory disturbances.

Important symptoms: Paralysis, Fibrillary Contraction and Muscular Atrophy.

The principal symptom in this case is a motor paralysis. We turn, therefore, to chart X. The paralysis certainly is a continuous one and of the three alternatives next offered us we must select the third, inasmuch as we have a flaccid paralysis with muscular atrophy in the head and arms and a mild spastic paralysis in the legs. We turn, therefore, to chart Xc. In this case the cranial and spinal nerves are involved, next there are no sensory symptoms, next the disease is chronic, and finally the lips, tongue, larynx and pharynx are involved; consequently the diagnosis is Progressive Bulbar Paralysis. But this diagnosis does not explain the paralysis and atrophy of the muscles of the shoulder which are supplied by spinal nerves. We turn, therefore, to the next sub-division, where spinal nerves are alone involved, and follow through, no sensory symptoms and through a paralysis involving the shoulder girdle muscles, and reach the diagnosis of Amyotrophic Lateral Sclerosis. The diagnosis is, then, a combination of two diseases: Progressive Bulbar Paralysis and Amyotrophic Lateral Sclerosis, and we find in the abstracts of these diseases that they often occur together in combination.

If we consider the symptom "fibrillary contraction" with the aid of chart XIIb, it is evident that this is an organic and not a functional disease, that there is a marked muscular atrophy and that there are no sensory symptoms, and thus the diagnosis of both Progressive Bulbar Paralysis and Amyotrophic Lateral Sclerosis is confirmed. Finally if we consider the symptom "muscular atrophy" with the aid of chart XVIIa we find that the atrophy is considerable and of a relatively rapid course, that there are no muscular hypertrophy and no sensory symptoms and thus we are led again to the same diagnosis,

Case VI. Male, aet. 12.—During the first year of his life the child had great difficulty in retaining food. At the end of his first year he began to have convulsions with unconsciousness, and, with the exception of an interval of two years, these have continued up to the present time; the last attack having occurred three weeks ago. The child has a very small head and an idiotic expression of face. He apparently understands most of what is said to him, but he can talk only a very little and only a few words are intelligible. There are no contractions or deformities, and he uses his arms and legs well.

Important symptoms: Anarthria and Idiocy.

The most striking symptom in this case is that a boy of twelve years can scarcely speak intelligibly. Turning to the table of contents we find that disorders of speech are treated in chart XIII, to which we turn. The loss of speech in this case is so nearly complete that it can

be called anarthria, which is discussd in chart XIIIa. The disease is evidently congenital, and the expression of the face is idiotic, and reading and writing are impossible; so that the diagnosis is Idiocy. Had we, on the other hand, decided that the child could speak, but very imperfectly and unintelligibly we should have sought for the disease in the same chart XIIIa, under the heading of dysarthria. Here the congenital nature of the defect and the absence of cleft palate, etc., would have led us directly to Imbecility. In order to trace the case further let us follow the cross reference after idiocy which is 1081 and which we find in chart XVIc. This case on account of his convulsions might be classed under Epileptic Idiocy or on account of his small head under Microcephalic Idiocy, or under both.

Case VII. Female, aet. 53.—Complains of trembling and that she cannot execute any movement quickly, because her arms and legs are stiff and rigid. When walking she has a decided tendency to pitch forward. Feels warm at times when the room seems cool to others. Expressionless face, passive tremor of hands. Propulsion and retropulsion when walking or standing. Rigidity of arms and legs. Difficulty in rising from a low chair. Knee-jerks rather increased.

Important symptoms: MUSCULAR RIGIDITY, TREMOR and ABNORMAL WALK.

The most characteristic symptom in this case is the rigidity of the arms and legs which is a mild tonic spasm. From the table of contents we learn that diseases causing spasm are treated in chart XI, to which we turn and find that general tonic spasm is discussed in chart XIb. In this case there is no fever and of the five sub-divisions under this head, this case clearly falls in the second: "rigidity which does not prevent passive or voluntary motions." Of the two alternatives next offered it is evident that we must choose the second, in the abstract of which we find all the symptoms present in our case. The diagnosis is, therefore, Paralysis Agitans.

If we follow the symptom "tremor," we find this treated in chart XIIb. It is a passive tremor and, whether it be increased or diminished on voluntary movements, if it be slow, the abstracts show that it is a case of Paralysis Agitans, because the other abstracts do not fit this case at all.

If we consider the difficulty in walking in this case we turn to chart XIIIc. In the three great divisions offered this case evidently falls in the third: "paralytic and spastic;" and of the two sub-divisions next offered we must take "general rigidity" which leads us again to the diagnosis of Paralysis Agitans.

Case VIII. Male, aet. 59.—During the past 34 years has had at times attacks of asthma. During the past four years has been troubled by a great many paroxysmal attacks of vertigo, at irregular intervals; some are slight, some are so severe as to throw him from a chair half way across the room to the floor, where he must lie for several hours, because when he raises his head from the floor he vomits violently and the dizziness becomes worse. He often has slight attacks of vertigo, which make him stagger when walking. During these four years he has been slowly growing deaf in his left ear; the deafness being now extreme. He has also had during the same time in the same ear, a buzzing and a ringing which is most intense just before an attack of vertigo. He has no paralysis and no loss of muscle sense. Bone conduction is absent. He also is much troubled by gastric flatulence, to which he attributes his vertigo, but when he takes digestive medicine and the digestive disturbances are relieved, the vertigo remains unchanged. His eyes were found to be astigmatic and proper glasses used, but no improvement in the attacks of vertigo followed.

Three years after the above record was made the attacks were milder and less frequent, but at that time his left ear was totally deaf and deafness was advancing in his right ear.

Important symptoms: PAROXYSMAL VERTIGO, STAGGERING WALK and DEAFNESS.

In this case the principal symptom is paroxysmal attacks of vertigo; diseases causing which, we learn, are treated in chart XVd. We see from this chart that vertigo may be caused by digestive disturbances and disease of the eye, both of which were present in this case, but the vertigo persisted when these abnormal conditions were relieved; so that they could hardly be the cause. On the other hand, we find that vertigo is associated with deafness, a prominent symptom in this case, and in looking over the abstract of this form of vertigo we see that it fits the case exactly; so that the diagnosis is Ménière's Disease.

If we consider another symptom, "the occasional staggering in walking," we find this treated in chart XIIa. This patient has no loss of muscle sense and no muscular paralysis. He is an adult and his hearing is abnormal and thus we are led again to Ménière's Disease. Finally if we trace the symptom "deafness" with the aid of chart XIVe we find that the deafness, at any rate at first, was unilateral, that bone conduction is absent, that there is no facial paralysis and that severe paroxysmal vertigo and tinnitus aurium are present; thus confirming again the diagnosis.

Case IX. Female, aet. 17.—Heredity good. Was well until about three years ago when, at the time of commencing menstruation, she began to have attacks of clonic convulsions with unconsciousness, which have continued up to the present time and in which she has occasionally bitten her tongue. Has also lesser attacks of unconsciousness, or very cloudy consciousness, in which she automatically prays, or says foolish things. Has no memory of any of her attacks. She has an immediate aura of fire before her eyes and of wheels revolving in her head. Some headache follows the attack. The convulsions occur only, and the lesser attacks mainly, at night. Physical examination is negative, urine normal. Fundus of eye normal. Knee-jerks equal. Much acne on face.

Important symptoms: Coma and Convulsions.

The constant symptom in all her attacks is unconsciousness, or coma, of short duration. This symptom is treated in chart XVIa. There is no history of recent injury, of brain disease, of poisoning, of heart disease. of paralysis, of kidney disease or of fever. Therefore, we are led at once to the diagnosis of Epilepsy or of Eclampsia. The latter can be excluded by the frequently recurring attacks at long intervals.

If we next take the symptom of clonic convulsion with the aid of chart XIa, we find that there is no fever and the convulsion is a universal one, and not local at the onset. There is coma and there are no symptoms of disease of the brain or cord, or of the kidneys, heart or blood, or of poisoning and thus we confirm the diagnosis of Epilepsy.

Case X. Female, aet. 34.—Nine years ago one morning, her left arm, leg and side of face felt numb and she could not see things on her left side without turning her head. These symptoms steadily increased during the day and she could not use her leg, and especially her arm, well. She could always walk, but at first she could walk only with difficulty. This difficulty in walking gradually passed away. She could use her arm, but could not use it well for more than a year, and it is not quite right even at the present time. The numbness of the left side and the inability to see things on her left still persist. On physical examination there are found anesthesia and analgesia of the left arm and leg and left side of body and face, (left hemianesthesia and hemianalgesia), also blindness in each eye for all objects to the left of central vision (left homonymous hemianopia). The left arm and leg are a little awkward and a trifle weak; strength of left hand grasp to that of right is as 80 to 105. Knee-jerks lively, perhaps stronger on left side: neither ankle-clonus nor Babinski. Organic reflexes normal.

Important symptoms: Hemianesthesia, Hemianalgesia and Homonymous Hemianopia.

The principal symptoms of this case are hemianesthesia, hemianalgesia and homonymous hemianopia. These are sensory symptoms and indeed, symptoms of a diminution of sensation, We turn to the table of contents and find that "diseases causing a diminution of sensation" are considered in chart XIV, which we next consult. Starting with disorders of sensation in the first column, we have five alternatives offered us in the second column, among which we ought, without doubt in this case, to select diminution of sensation and following this division we have in the next column three alternatives, among which, undoubtedly, we should select anesthesia and analgesia and turn to chart XIVa. In this chart we have the alternatives of the tendon reflexes being either absent or present. In the above case they are present. The dilemma in the next column is quickly decided because the organic reflexes are normal. The history of a motor paralysis lasting a year or more and still slightly persisting directs us to the first alternative in the next column, especially as there are no hysterical symptoms present; while the unilateral nature of the symptoms and next the acute onset (one day) brings us to the diagnosis of Cerebral Hemorrhage or Softening.

To determine which lesion is present, we follow the first cross reference, No. 503-6, which we find in chart Xb. In looking over the abstracts differentiating cerebral hemorrhage, embolism and thrombosis, our case, with its relatively slow onset, its absence of any coma, its absence of any source for an embolism, is probably one of cerebral thrombosis and certainly one of cerebral apoplexy.

The next question is as to the locality of the softening. To ascertain this we turn to the table of contents and find that "localization from symptoms of paralysis" is discussed in chart XXII to which we turn. The reflexes being present in our case, we are brought to the question: whether sensory or motor paralysis is dominant. In our case sensory paralysis is dominant and we turn to chart XXIIc. Of the first alternative offered us in this chart we must choose the first: anesthesia and analgesia. In regard to the next column, the distribution of the anesthesia in our case evidently falls into the class: "the (left) arm, leg and face are anesthetic." In our case there is no Jacksonian epilepsy and there is hemianopia, so that the localization of the softening is in the posterior part of the right internal capsule. If we now turn to Fig. 17 we can easily see how a lesion in the posterior portion of the internal capsule can easily involve the sensory fibers from one-half the body and also the optic fibers; the continuation of the optic tract. It is also easy to understand that on account of the wide-spread circulatory disturbances in the early stages of the disease, before a collateral circulation had, to a degree, reestablished itself in the periphery of the lesion, the motor fibers lying directly anterior should be involved and a more or less temporary hemiplegia should occur, as was indeed the case. It might seem strange that deafness did not occur in this case as it is certain that the auditory fibers also must have been involved in the lesion, but it is well known that central lesions only produce deafness, even unilateral deafness, when the lesion is bilateral (see page 8.)

Thus we have arrived by means of the charts to the diagnosis of this case of "thrombosis of the artery supplying the posterior portion of the internal capsule," but in order to make this diagnosis doubly sure, let us take another one of the prominent symptoms, such as homonymous hemianopia, and follow it through the charts. This symptom is also a diminution of sensation and therefore we turn again to chart XIV. Disregarding this time diminution of sensation we follow "disturbances of vision" and "limitation of field of vision" to chart XIVb. Here we find homonymous hemianopia and in the next column there can be no doubt that we must choose the path which hemianesthesia indicates and by it are led to the diagnosis of hemorrhage, or softening, in the posterior part of the posterior limb of the contralateral internal capsule. which is the diagnosis which we had already reached by another road.

CHART X

Motor Paralysis

DIAGNOSTIC ANALYSIS OF SYMPTOMS.

Symptoms Analyzed	Tests: Permanence of Paralysis	Tests: Reflexes in Paralyzed Muscles	
469 MOTOR PARALYSIS OR PARESIS (244) After a careful examination has shown that the paralysis is a true one and is not simulated by any ankylosis or by pain on motion.	470 CONTINUOUS PARALYSIS	The reflex acts in the paralysed muscles are absent or decidedly diminished.	
		472 FLACCID PARALYSIS Lesions of peripheral motor neurons. There are hypotonia and changes in the electrical reaction of the nerves and muscles involved in very varying degree from simple diminution in excitability to complete reaction of degeneration. No associated movements present.	The differential diagnosis of those diseases in which FLACCID PARALYSIS occurs is set forth in CHART X a.
		The reflex acts in the paralysed muscles are normal or exaggerated.	
		473 SPASTIC PARALYSIS (251) Lesions of central motor neurons. There is hypertonia without alternations of electrical reaction of the nerves and muscles. Associated movements (synkineses) may be present.	The differential diagnosis of those diseases in which SPASTIC PARALYSIS occurs is set forth in CHART X b.
		474 A combination of FLACCID PARALYSIS in the upper part of the paralyzed area and of SPASTIC PARALYSIS in the lower part.	The differential diagnosis of those diseases in which there is a combination of FLACCID and of SPASTIC PARALYSIS, and of those in which INTERMITTENT PARALYSIS occurs is set forth in CHART X c.
	471 INTERMITTENT PARALYSIS.	All the muscless of the body and head. The muscles of one or both legs, rarely of arms. Commencing in legs, extending to arms. Associated with a cervical rib.	

CHART Xa

Flaccid Paralysis

Comprising Numbers 475 to 477 on left side of Chart
and 482 to 500 on right margin

472 FLACCID PARALYSIS

- **475** — No muscular atrophy, except rarely in chronic cases. Reflexes may be diminished only, not abolished.
  - Paralysis beginning in the feet and ascending to the head in *adults*. — The paralysis is in the form of a paraplegia, commencing in the producing bulbar symptoms (424), and causing death usually i sensory symptoms are pronounced the disease is probably a neu sembling those of Landry's paralysis and in addition hematoporphy
  - No true paralysis but great hypotonia in *infants*. — Occurs usually congenitally, rarely during the first year of life. N be placed in very abnormal positions. The child cannot use the sli disturbances, no disturbances of organic reflexes. Electrical reacti
- **476** — Muscular atrophy, usually great, following the paralysis after the second week of the disease.
  - Paralysis primary.
  - The Degenerative Atrophies. (See also Syringomyelia —553, 840-1.)
  - The organic reflexes are permanently disordered. (1, 324-5.) — Marked sensory symptoms, such as pain, paresthesiae, anesthesia, etc., are present with the motor symptoms. The legs only are paralysed and exhibit trophic disturbances. There is incontinence of urine and the bladder is empty or nearly so. Defecation is usually disordered.
    - Very acute onset. Sym hyperesthesia. May
    - Acute, sub-acute or chr history or other evide
    - Very chronic and progr tative symptoms, intr
    - Very acute, acute or su symptoms less symm normal. The anesthe the lesion. Dissociati
  - The organic reflexes are normal or show only transitory disturbances (1 and 323-4.)
    - Sensory symptoms, such as pain, nerve and muscle tenderness, paresthesiae, anesthesia, etc., are present. In very exceptional cases sensory symptoms may be practically absent.
      - Many spinal (very rarely cerebral) nerves are affected. — There is usually a hist acute, sub-acute and and arms (long neur weakness, atrophy an duction of pain and l form of Korsakow's form rarely runs an s form exhibits many s
      - The paralysis is coincident with the distribution of one, rarely of a few spinal nerves. — The motor and sensory paralysis (anesthesia) may be slight and the pain great. The motor paralysis is usually greater than the sensory.
      - The paralysis is confined to the distribution of one or more cranial nerves.
        - Occurs most commonly paralysed. For speci
        - The paralysis is usually
      - Extensor muscles are alone affected. — Usually confined to the lead poisoning. Prev
    - No sensory symptoms, except rarely pain and tenderness over the nerve trunks in early stage.
      - A paralysis of acute onset, usually confined to the arms and legs, generally to a portion of one or both; in rare cases involves the cranial nerves. A chronic form may occur exceedingly rarely. — The paralysis (which is sions. Rarely there i so in children. The p In infants and young neuritis with predomi toms are common an of functionally related muscles of the trunk and scurvy (Barlow's form, (4) encephalitic pathogenesis. Chronic it is certainly very r
      - A paralysis of chronic onset commencing in the peronei muscles and extending symmetrically. Intrinsic muscles of the feet affected. — T
- **477** — A combination of muscular atrophy, and apparent hypertrophy.
  - Paralysis secondary.
  - The Muscular Dystrophies. (107) — A chronic disease commencing in childhood or youth and usually showing marked heredity. In rare cases it may commence in middle life, or even later. It exhibits a progressive muscular atrophy, usually combined with some hypertrophy, hence called muscular dystrophy. In time all the muscles become atrophied. The organic reflexes are normal and there are no sensory symptoms whatever and no motor paralysis, except such as would result from the muscular degeneration. Even the apparently hypertrophied muscles are weak. Tendon reflexes are early much diminished and finally absent in the affected muscles. There are no fibrillary contractions. The course of the disease is progressive, but very chronic, lasting many years. From its point of commencement the atrophy extends throughout the body. It produces a marked lordosis. Although the muscular dystrophies are divided into three groups, there are many transitional and mixed forms, and the examination of the excised muscles also shows mixed forms.
    - The disea extremi muscles with a trophy ent part More or
    - The disea tremitie cles sho mixture and esp terstitia

CHART Xb

Spastic Paralysis

Comprising Numbers 478 to 481 on left side of Chart
and 501 to 527 on right margin

473 SPASTIC PARALYSIS

- **478 Hemiplegia or Diplegia or Monoplegia (254-5, 258)** — Congenital or acquired in infancy. There may be fever or apoplectic symptoms at onset. — A motor paralysis of one (infantile hemiplegia) or both sides (Little's are common and may mask the exaggerated reflexes. In walking ther and at times idiocy or insanity. Frequently there is partial arrest of cerebral diplegia, bulbar symptoms (424) are present without fibril expressions, etc., can occur involuntarily, but no voluntary motions.
- **479 Hemiplegia, or Monoplegia (254, 258) (See also Syringomyelia—553, 840-1.)**
  - Almost always in adults and after middle life. Sudden onset, or stroke (ictus), usually with coma (205 and 1037), or with headache or vertigo and mental confusion. Not infrequently the attack commences with a hemiplegia which may or may not be followed by coma.
    - Symptoms of irritation (convulsions, rigidity, etc.) are more pronounced than are symptoms of paralysis. — The disease which is Cerebro-s
    - Symptoms of paralysis are more pronounced than those of irritation (convulsions may occur, especially in cortical lesions and in hemorrhage into the ventricles, in which case lumbar puncture may yield a bloody fluid.) The paralysis is in part temporary and in part permanent in varying degree. Slow improvement with almost perfect recovery in rare cases. More or less permanent mental impairment, often very slight. Usually patients are more emotional than previously. Exaggerated reflexes and ankle-clonus are present after coma has cleared up. Babinski is present from the start. Puffing, stertorous respiration is common. Cheyne-Stokes respiration (425) and tracheal rales are very unfavorable symptoms. Certain muscles are more frequently and severely paralysed in apoplexy than others. Wernicke's predilection muscles (254).
      - A sudden a lysed tha tion of ey often art face, ther hemiplegi nicke's pı
      - Similar to arterial t mon than
      - Similar to syphilis. basilar a hemorrha
  - Gradual onset without coma, except as a terminal symptom.
    - Sensory symptoms are always present. Organic reflexes are normal or only slightly disordered.
      - Brain symptoms. Steadily increasing psychic disorder, and local motor and sensory disturbances over the same area. — Headaches, common. ent in tu inertia a spasmodi epilepsy fluid and
      - Spinal symptoms. Paralysis of motion and sensation on opposite sides of body. — Characteris course, s spinal ep
    - Choreic symptoms. — The paralysis is only slight and follows or accom a paralysis (chorea mollis).
- **480 Paraplegia (257) (See also Syringomyelia—553, 840-1.)**
  - There is paralysis always of motion and commonly of sensation, usually in the form of paraplegia, more rarely in the form of a spinal hemiplegia (432), which later may become a paraplegia. The reflexes are exaggerated. Ankle-clonus and Babinski are present. Spasms and contractures and bed sores are often present. The motor paralysis is permanent or lasts a very long time. Sensory paralysis may be slight and transitory and may be altogether absent. The anesthesia is often limited above by a narrow zone of hyperesthesia.
    - Cranial and spinal nerves are involved. — Intention tremor, nystagmus, scanning speech, a
    - Arms and legs are paralysed. Priapism is common, also respiratory difficulty and early death. Radiating pains are common.
      - There may be a history of injury and a fracture
      - No history of injury. Little or no pain. Senso litic myelomalacia (1211).
      - May be history of remote injury. Much pain radi fluid. In early stages, extra-medullary tumors
    - Legs only are paralysed. Girdle sensation and pains radiating into the extremities are common.
      - There may be a history of injury and a fracture
      - No history of injury. Little or no pain. Senso myelomalacia (1211).
      - May be a history of remote injury. Much girdle extra-medullary tumors produce irritative, intr
      - Evidence of Pott's disease or tumor compressing pain. In cases of compression due to Pott's di increased tension and may contain globulin an
      - History of working under increased atmospheric
      - Old age, atheromatous arteries, arterial tension
      - Tumor can be seen or felt on back replacing th or not. Club-foot is common.
      - Signs of irritation predominate over those of pa unless the cord is involved. Usually a history
    - Legs mainly involved. Arms involved later and slightly, if at all. These diseases may occur in severe anemia.
      - No ataxia. — Paralysis purely motor, a paresis to passive motion, especially wh of a multiple sclerosis (659). E its etiology. This disease may be
      - Ataxia. — There is a combination of motor p and Babinski are present. In so
- **481 Paralysis of any extent: *local, monoplegia, hemiplegia, or paraplegia*** — Paralysis limited by some prominent *anatomical landmark.* — The motor paralysis is usually accompanied by a great variety of sens physician (imaginary or delusional paralysis). A paralysed limb oft duration. Organic reflexes rarely disturbed, but retention of urine is ments (436)) do not occur in hysterical paralysis, and this is somet In walking sideways, stepping laterally along a straight line, in hyst

CHART Xc

Combined and Intermittent Paralyses

Comprising Numbers 471 and 474 on left side of Chart
and 535 to 557 on right margin

474 COMBINED PARALYSIS

- **Cranial nerves alone involved. (Figs. 18, 23, 33, 38.)**
  - Sensory symptoms present usually.
    - Bilateral symptoms. — If the patient does not promptly die, one or more cr gia (525). There are usually dysarthria, dyspha toms at first may be more unilateral.
    - Crossed paralysis (256) and bulbar symptoms (424).
      - Paralysis of one or more eye muscles of one side and
      - Paralysis of facial (both upper and lower branch
      - Paralysis of hypoglossus of one side and of arm an
  - No sensory symptoms.
    - Acute
      - A disease caused by eating spoiled food (sausage, even aphagia. The most characteristic symptom i especially the externus, are paralysed. At the aut pation is a prominent symptom. The disease has
      - The onset of paralysis is sudden. If the patient gressive. They are usually unilateral, but may b a spastic paralysis more or less pronounced in th flammation, hemorrhage, thrombosis, embolism, o syphilitic endarteritis or syphilitic neuritis, or t
    - Chronic—The chronic forms of these diseases, with the spinal form (547-8) constitute the progressive muscular atrophies and resemble the muscular dystrophies in that the paralysis and atrophy advance together slowly, and it is difficult to say which is primary. They also constitute a group of chronic degenerative atrophies. The paralysis is purely motor.
      - The paralysis involves the eye muscles. — A grad mobi plegi
      - The paralysis involves the lips, tongue, pharynx and larynx. — The he uvul A di there more
- **Spinal nerves alone involved (Figs. 24-7, 33, 38.)**
  - No sensory symptoms.
    - Symmetrical paralysis commencing in the small muscle of hands or in shoulder girdle muscles.
      - The muscles affected show progressive weakness, bands, or, more rarely, in the muscles of the sho atrophy. The thumb cannot be brought across h Babinski, but not always. There are secondary al irritability of muscles is increased. Often ass eral columns are involved or not. It is difficult
    - Symmetrical paralysis commencing in the muscles of the thighs and buttocks.
      - Same symptoms as above, but without spastic, or o
      - This rare disease commences, in the first or second end of a few years. The muscles exhibit the re tory lesions and without any involvment of the
  - Marked sensory symptoms are present, such as pain, paresthesiae, anesthesia, etc., with the motor paralysis.
    - Both arms and legs are paralysed. There are trophic disturbances in the arms and not in the legs. Pupils are often unequal. Reflexes are abolished in the arms and increased in the legs. Babinski and ankle-clonus are present. The bladder is usually more or less distended; its detrusor being paralysed. Contractures may be present in the legs.
      - Very acute onset. Symptom may yield a bloody fluid.
      - Acute, sub-acute or chronic litic myelomalacia (1211).
      - Chronic course, intense pain tensors and, in the later s phocytosis. Most of these
      - Very chronic onset and a p irritative, intra-medullary
  - Dissociation of sensation (365) is present.
    - Dissociation of sensation is the most characteristic symptom and is com Trophic lesions are usually prominent. Pemphigus, ulceration and mu trophic symptoms predominate over motor symptoms in the arms; wh present the symptoms may be both in arms and legs, and the motor sy gerated in central gliosis in the cervical or dorsal regions. The cervic rapid course, and may exhibit a unilateral, spastic, muscular paralysis

471 INTERMITTENT PARALYSIS

- All the muscles of the body and head.
  - The characteristic sign of the disease is the rapid tiring of the muscles when in action. Pat action (399). There is no muscular atrophy and no reaction of degeneration. In the domai is usually held retracted on account of the ptosis. The symptoms are slight in the mornin rhagia) in the muscles at the autopsy.
- Muscles of one or both legs, rarely of arms.
  - Intermittent attacks of painful muscle cramp, and weakness of leg or legs, caused by walking. Rarely the disease occurs in one or both arms. No sensory disturbance except painful cr
- Commencing in legs extending to arms.
  - Recurrent attacks of paralysis of the muscles of the legs usually first and then of arms, lastin nerves are not attacked. There is usually well marked heredity, or the disease occurs in fa but in some groups of family periodic paralysis these negative symptoms are not present.
- Associated with a cervical rib.
  - A cervical rib can be felt and can be seen with the X-ray. In some cases of cervical rib, at the skin which comes on after the arm has been used a short time, and, if use of the arm is ralgia, may be caused by a cervical rib, and is often relieved by elevation of the arm an
- *Associated with arterial disease.*
  - *Repeated attacks* of temporary paralysis are very rarely met with in hysteria and in elderly p of cerebral origin such paralyses are of limited extent; when of spinal, general. See also

CHART XI

Convulsions and Spasm

DIAGNOSTIC ANALYSIS OF SYMPTOMS

Symptom Analyzed	Tests: Character	Tests: Extent	
570 CONVULSION OR SPASM (242)	571 CLONIC mainly (246)	GENERAL CONVULSION	Diseases in which convulsions occur are set forth in Chart XIa.
		LOCAL CLONIC SPASM	Diseases in which local clonic and all forms of tonic spasm occur are set forth in Chart XIb.
	572 TONIC mainly (245)	GENERAL TONIC SPASM	Diseases in which local clonic and all forms of tonic spasm occur are set forth in Chart XIb.
		LOCAL TONIC SPASM	Diseases in which local clonic and all forms of tonic spasm occur are set forth in Chart XIb.
	573 CHOREIFORM (272)		Diseases in which choreiform and athetoid spasm occur are set forth in Chart XIc.
	574 ATHETOID (271)		Diseases in which choreiform and athetoid spasm occur are set forth in Chart XIc.

CHART XIa

General Clonic Convulsions

Comprising Numbers 571 on left side of Chart
and 575 to 596 on right margin

Idiopathic epilepsy—575
Symptomatic epilepsy—576 to 596

DIAGNOSTIC SYMPTOMS AND TESTS

571 GENERAL CLONIC CONVULSION

- Apyrexia.
  - The convulsion commences in all the muscles at about the same time (epileptiform convulsion).
    - Loss of consciousness, (coma or semi-coma) (205). Frequently biting of the tongue or other injury. Short duration.
      - No other symptom of disease except the convulsion.
        - Repeated attacks. — Th
        - One attack or one series of attacks. — A
      - Symptoms of serious brain disease.
        - Congenital or in infancy. Often fever at onset of first convulsion. — Th
        - In youth or more often in adults. — Re
      - Symptoms of cerebro-spinal disease. — In
      - Symptoms of disease of other organs than the brain.
        - Kidney disease. — Th
        - Cardiac disease. — Sl
        - Blood disease. — Ra
      - Symptoms of poisoning.
        - Blue line on gums, lead
        - Alcoholic odor of breath
    - Apparent, but no true, coma (shown by susceptibility to suggestion during the attack). No biting of tongue or other injury. Long duration.
      - Symptoms of hysteria (415). Such attacks have been called hystero-epilepsy. — Th
  - The convulsion always commences in one group of muscles and later extends over the whole or part of one side of the body and often over both sides. Jacksonian epilepsy (421).
    - If the convulsion remains unilateral, consciousness may or may not be lost, usually not, but it is always lost when the convulsion becomes bilateral. — Th pr of di al
- Hyperpyrexia.
  - Epileptiform convulsion.
    - Coma during and after the convulsion. — Oc
- Pyrexia. See also 577.
  - Epileptiform convulsion. Coma during and usually after the convulsion.
    - Headache, backache and radiating pains, delirium, vertigo and vomiting, especially on change of posture, hyperalgesia (spinal and elsewhere), photophobia, etc., are early symptoms. Retraction of head, opisthotonos, etc. (285). Paralysis of cranial nerves (squint, etc.), cutaneous eruptions (herpes), tâches cérébrales and Kernig's symptom (320): Tonic spasm and paralysis are more common in basal inflammations, and clonic spasm in cortical inflammations.
      - Lumbar puncture gives a bloody or purulent, f creased pressure cont and many polymorph cytes.
      - Lumbar puncture gives a c increased tension and c ulin and many monon cytes, and if the dis polymorphonuclear leuc
      - Lumbar puncture gives a c increased tension, but cellular elements.
  - Coma during the convulsion.
    - May occur in children at the onset of any infectious disease, especial
    - Is the result of some unusual metabolic changes within the body, a

CHART XIb

Clonic or Tonic Spasm

Comprising Numbers 571 (continued) and 572 on left side of Chart
and 597 to 621 on right margin

(Note)—Many of the spasms, especially the tonic spasms, are associated with pain, and are then called "cramps."

DIAGNOSTIC SYMPTOMS AND TESTS

571 CLONIC SPASM

- **LOCAL CLONIC SPASM**
  - **Pyrexia in very acute cases.** — Shock-like spasms similar to that produced by an electric shock. — Begins in one arm and si side and then to oppos
  - **Apyrexia.** — A single or many times repeated spasm, rarely contracture, of one muscle or of a group of muscles, occurring in paroxysms which rather tend to subside on voluntary movements. Myoclonus (270).
    - Occurs in face and more rarely in neck and arms. — The a T t
    - Begins in arms and may extend to legs, but almost never to face. Often the tendons play as in subsultus tendinum. Symptoms show considerable variation in different cases. In rare cases there may be infection, fever and delirium (Hunt). — The n a a n
    - Begins in side of face or in one arm or leg and may extend over one, or even both sides of body. — The f

572 TONIC SPASM

- **GENERAL TONIC SPASM**
  - **Pyrexia.**
    - **Spasm commencing in jaws.** — There is the history of an infected wound, or s jaws, occurring in paroxysms; also rigidity c being held in position of opisthotonos, emprc very high. The disease varies greatly in inte
    - **Spasm commencing in pharynx and oesophagus.** — There is a history of a bite by an animal (usu especially on sight of water. Spasmodic cl cough, opisthotonos and general spasms are light and accommodation. The stage of exci nosis must be made in such cases by the pre
    - **Spasm commences in back of neck and in back.** — There may be more or le tom. Lumbar puncture
  - **Apyrexia. If unconsciousness is present, see also epilepsia media (575).**
    - **Rigidity of spine.** — May follow injury. Blood in cerebro-spinal fluic
    - **Cerebellar ataxia is present (281).** — A tonic spasm of sudden onset, the face not being affected.
      - Th t
      - Re
    - **Rigidity rather than spasm, not strong enough to prevent passive or voluntary movements (266).**
      - Extremities and trunk r last minutes, hours or 1047.
      - Rigidity of all muscles.
    - **Spasm only at commencement of any action.** — Spasm passes away as the action is continued, the muscles of the face usually escape altoget show marked hypertrophy. Closely allied to exposure to cold with consequent reflex vaso- so-called acquired form, "myotonia acquisita
    - **Spasm mainly confined to hands and feet, paroxysmal.** — Bilateral painful tonic spasm of muscles of han Increased mechanical (Trousseau's phenome rickets or digestive disorders and intestinal infectious diseases, in poisoning and in preg
    - **General painful clonic, followed by tonic, spasm.** — Spasm very general and poisoning. Death usua
    - **General permanent contracture.** — Paralysis is coincident v
- **LOCAL TONIC SPASM**
  - **Apyrexia.**
    - **Spasm only occurs when performing some accustomed act.** — Occurs usually in small muscles and in those gradual onset, steadily grows worse, and rend rather than spasm. Atrophy of the muscles of singers, public speakers, etc.
    - **Rather brief spasm of one or more muscles.** — A spasm lasting minutes or hours, due to local urethral spasmodic stricture, vesical spasm,
    - **Tonic persistent spasm.** — A spasm of varying intensity attacking an arm occurs.
    - **More permanent spasm.** — A contracture of a few or many muscles usua efforts are made to overcome it. No muscle (674) or may consist in jumping or skippin
    - **A permanent or, at least, a long continued, contracture. The muscles are anatomically shortened in later stages.**
      - **A hemiplegic contracture.** — Tendon reflexes are incre lasts for years. Usual
      - **A paraplegic contracture.** — Tendon reflexes are incre an attack of parapleg
      - **A local contracture.** — Absence of reflexes. Mu of tendon and fascia

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CHART XIc

Choreiform and Athetoid Spasm

Comprising Numbers 573 and 574 on left side of Chart
and 622 to 632 on right margin

		DIAGNOSTIC SYMPTOMS AND TESTS		
573 CHOREIFORM SPASM (113, 126, 272)	GENERAL	TRUE CHOREIFORM MOVEMENTS	Widespread spasmodic contractions of muscles of body generally.	Irregular, quick, involuntary, spontaneous contractic then of another group of muscles throughout the bo half of the body (hemichorea). Patient is restless is explosive. Sounds are often made involuntarily. ' somewhat the character of purposeful movements, closely extreme restlessness; grimaces, thrusting ou of hands and feet, etc. Some muscular weakness 510) is present; and almost always marked hypot 252, 472). Voluntary movements are interfered v ordinate by the occurrence during them of these i tions (ataxia). The part cannot be held still. ' ments may be slight, or so strong as to prevent v speaking. They cease during sleep, but to some e They are worse under observation and excitement. mal but the knee-jerk may be protracted and the slowly. Paresthesiae and anesthesia rarely present. dence that chorea may be due to disease of certain c destructive lesions of the caudate nucleus and of lenticular nucleus produce the symptoms of chorea
			Limited to one group of muscles.	Involuntary, often unconscious, or unnoticed, executi coughing, hemming, winking, etc. Each person ha
		PSEUDO-CHOREA	Sudden, lightning-like contractions of groups of muscles.	The spasms are instantaneous; the platysma, stern especially in Northern Italy. In the later stages, ably not at all related to chorea minor but is more
			A coarse tremor rather than choreiform movements.	Usually limited to one extremity. Rhythmical trembling of an ex times more like electric shock times called chorea magna or
574 ATHETOID SPASM (271, 503)	LOCAL	A slow contraction of one set after another of small muscles of the hand; rarely of the foot (mo and wrists frequently also involved and are usually held contractured in extreme flexion. Squi tion of fingers, extension and hyperextension predominating. The athetoid spasm is increased ments of the same or of the other hand. The face and neck muscles may not infrequently be athetosis. The extremity or extremities involved are always weak but never paralysed. The sp eral or bilateral. Usually in hands, more rarely in feet. These movements, though slow, are pc cause subluxation of joints. Decided muscular rigidity is usually present.		

OF SYMPTOMS

HETOID SPASM

?MPTOMS		DIAGNOSIS	
:t of one and limited to one-dgety. Speech ovements have resemble more ›ague, twisting :ic paralysis—or atonia (39. nd made inco-ntary contrac-choreic move-g or eating or prevent sleep. lexes are nor-tak back only re is some evi-lar tracts, and utamen of the	Common in children, rare in adults. Slight mental disturbances often present. Usually acute, rarely chronic, frequently recurrent. Often associated with rheumatism and endocarditis, rarely with pregnancy (chorea gravidarum). The prognosis is good, but in pregnancy is serious and the uterus should be emptied.	Sydenham's, or Infectious, Chorea. Chorea Minor (272).	622
	In some cases of chorea, the mental symptoms usually present in some degree, become extreme and dominate the clinical picture. In these cases the patients may exhibit hallucinations and a maniacal delirium, consciousness may be clouded and the intellect may progressively degenerate into dementia. This form of chorea runs its course with fever, is most common in adult pregnant women and frequently terminates fatally.	Chorea Insaniens.	623
	Occurs only in adults. There is much and progressive mental impairment. Movements coarser and more violent. Heredity. Chronic.	Huntington's, or Hereditary, Chorea (103).	624
	Occurs only in old persons with atheromatous arteries and brain symptoms. It is usually progressive and the mortality is rather high.	Senile, or Degenerative, Chorea.	625
	Occurs in hemiplegia, (after apoplexy, etc.) and is confined to the incompletely paralyzed extremities, especially the hand and arm. It is most frequent in the hemiplegias of childhood. Sometimes a prehemiplegic form is met with.	Post-hemiplegic Chorea, (501)	626
the same act at short intervals. Little "tricks" which characterize many persons such as own individual trick or habit and rarely varies from it. Usually occurs in neurasthenics.		Habit Chorea or Habit Spasm (274).	627
o-mastoid and hypoglossus muscles are especially affected. It is a rare disease, occurring ›tiform convulsions and paralyses with atrophy occur. Often fatal. This disease is prob-l to myoclonus (600-1).		Electric Chorea. Dubini's Disease (597).	628
:y, varying in intensity. At times so coarse and irregular as to resemble chorea, at other ther symptoms of hysteria present (415). The extensive convulsive movements some-›r (273) are purely hysterical and are not choreic in their nature.		Rhythmical, or Hysterical, Chorea (273).	629
›asm). Ankles twisting mo-luntary move-ed in bilateral nay be unilat-l and at times	The athetoid spasm is present from birth. It is very rarely unilateral, more frequently bilateral. There is much mental impairment, even idiocy.	Congenital Athetosis, (501).	630
	Present from birth or infancy. Some mental impairment. Unilateral or bilateral. Associated with a mild hemiplegia or diplegia. Rare.	Athetosis after cerebral palsy of childhood, (501).	631
	Occurs in adult life after an attack of apoplexy. Usually unilateral. Rare.	Athetosis after apoplexy, (503).	632

CHART XII

Perversion of Motion and Local Palsies and Spasms

DIAGNOSTIC ANALYSIS OF SYMPTOMS

Symptoms Analysed	Character	
635 PERVERSIONS OF MOTION (243)	638 ATAXIA (248)	The diseases in which ataxia occurs are set forth in Chart XIIa.
	639 TREMOR (250)	The diseases in which tremor, nystagmus, or fibrillation occurs are set forth in Chart XIIb.
	640 NYSTAGMUS (292)	
	641 FIBRILLARY CONTRACTION OR FIBRILLATION (293)	

LOCAL PALSIES AND LOCAL SPASMS

636 LOCAL PALSIES	See Chart XIIc.
637 LOCAL SPASMS	See Chart XIId.

CHART XIIa

Ataxia

Comprising Numbers 638 and 642 to 644 on left side of Chart
and 647 to 664 on right margin

638 ATAXIA (248)

- **642** Ataxia mainly upon standing or walking. Staggering gait. Static ataxia. Cerebellar ataxia. Asynergia major. (281).
  - No loss of muscle sense. No motor paralysis, except in late stage of 650-1.
    - Occurs at any age, usually in adults. Usually sensory symptoms.
      - Sight and hearing normal.
      - Sight or hearing abnormal.
    - Occurs in youth. No sensory symptoms.
      - Occurs in family group though less pronounced stagmus is common a
- **643** Inability to stand or walk. More or less complete.
  - Bilateral.
    - Many sensory symptoms.
      - Evidently functional.
  - Unilateral. (Hemiataxia.)
    - Often analgesia and thermic anesthesia.
      - Evidently organic.
- **644** Ataxia of all movements. Dynamic ataxia. Motor ataxia (280).
  - Unilateral. (Hemiataxia.)
    - Loss of muscle sense and sensory symptoms usually prominent. Knee-jerk usually increased.
      - When of acute course ally follows an apopl arterial disease is chronic course chol present.
  - Bilateral.
    - Knee-jerks normal.
      - No ankle-clonus. Hi
    - Exaggerated knee-jerks, ankle-clonus and Babinski.
      - Great variety of local intention tremor, sc Rarely the disease r is the presence of sy
      - A combination of symp usually lost before a jerks may be abolish
    - Knee-jerks and ankle-clonus absent. No Babinski. Often loss of muscle sense and retardation of conduction of pain.
      - Rarely any permanent are not uncommon e apart and feet are (438). Argyll-Robe and paresthesiae a and in cuirass, ulna fluid. The disease Wassermann also u affected. In the o diagnosis must rest
      - Slight motor paralysis normal. Cranial n never so chronic as
  - Irregular distribution.
    - Knee-jerks usually exaggerated, but no Babinski or ankle-clonus. Evidently functional (pseudo-ataxia).

CHART XIIb

Tremor, Nystagmus, Fibrillation

Comprising Numbers 639 to 647 on left side of Chart
and 666 to 697 on right margin

- **639 TREMOR (160-3-5, 250)**
  - **645 Intention Tremor (291).**
    - Coarse, irregular tremor; 4 to 8 per second.
      - Tremor is usually associated with scanning speech, n
        Usually a great variety of motor and sensory sympto
        sionally with their loss, over a very variable area. Th
        Vertigo is a very common symptom.
      - Symptoms resembling very closely those of multiple s
        resemble closely, if they are not identical with, dys
        lenticular nucleus and very commonly lesions of the
      - Occurs in family groups, but is neither hereditary nor congenital.
        - Occurs in athetosi autopsy
      - Occurs in family groups and shows well marked heredity. Staggering gait. Ataxia. Nystagmus is common and speech often defective.
        - Occurs af
        - Occurs be
    - Fine tremor.
      - Tremor is associated with general weakness or conva
    - Fine, rapid tremor; 8 to 12 per second.
      - Exophthalmos, goitre, tachycardia, vascular throbbing mally when patient looks downward (Graefe's symp
      - History of addiction to alcohol or drugs. Mental sym greatly at different times.
      - Presence of hysterical symptoms (415). Tremor is wo
  - **646 Passive Tremor. Increased on voluntary motion and excitement (290).**
    - Slow tremor; 3 to 6 per second.
      - Tremor is marked in face, lips and tongue; not uncon Lumbar puncture shows a lymphocytosis and globuli
      - Slow tremor of hand and foot of same side, associated
      - Tremor, which is associated with muscular rigidity an and finally involves the other side. The tremor is u stages. Characteristic attitude (head and body bent He has a similar tendency to run backwards (retro sine agitatione). The disease often commences with There are no sensory symptoms except the sensation argica (1047). Destructive lesions of the pallidal
  - **646a Passive Tremor. Diminished on voluntary motion (290).**
    - Slow, fine tremor; 3 to 6 per second.
      - Tremor begins bilaterally. Head is early affected. N
    - Slow, coarse tremor.
      - Rotatory or nodding tremor of head occurring sudden volved. The tremor ceases when the child's eyes ar
      - A series of jerky tremors limited to the back, or invol
  - Either Intention or Passive Tremor.
    - Not associated with other symptoms. Hereditary ba

- **640 NYSTAGMUS (292)**
  - Always a symptom of organic disease. Very rarely, an hysterical clonic spasm may simulate true nystagmus (pseudo-nystagmus). This is often vertical and is more rapid and more violent than nystagmus and is associated with other hysterical symptoms (415).
    - No weakness of any rectus muscle.
      - Impairment of sight.
        - Defective vision from whatever
        - Due to lack of pigment in iris,
        - Workers in mines. Due to wor
      - No impairment of sight.
        - Vertigo is a prominent symptom.
          - Paroxysm lently.
          - Vertigo, c
        - Coarse, jerky tremor is a prominent symptom. Ataxia is also present.
          - Occurs in
          - Occurs at
        - Cerebral symptoms present.
          - Occurs in
        - Rickety baby in winter.
          - The nyst nystagu
        - Congenital.
          - Lateral o
    - Weakness of one or more of the recti muscles.
      - Nystagmus occurs in convalesce

- **641 FIBRILLARY CONTRACTION OR FIBRILLATION (293).**
  - Evidence of organic disease. Degeneration of peripheral motor neurons.
    - Marked muscular atrophy with muscular weakness.
      - Marked sensory symptoms.
        - Analgesia
      - No sensory symptoms.
        - Muscular
        - Muscular
        - Muscular
  - Evidence of functional, not organic, disease.
    - No muscular atrophy or weakness.
      - Occurs u

CHART XIIc

Local Palsies

Comprising Numbers 700 to 721 on right margin.

(Note)—The anesthesia accompanying these palsies can be seen from the areas of cutaneous distribution of these nerves depicted in the plates at the end of the book (Figs. 33-8). In mild lesions of the nerves anesthesia is either absent or much less marked and less extensive than is the motor paralysis.

LOCAL PALSIES

Inability to Move, more or less, Muscles of the	Abstract of Symptoms	Diagnosis
EYEBALL	There are ptosis and strabismus divergens and the pupil is dilated and immobile both to light and accommodation (this condition of the pupil may occur as an isolated paralysis,—333). The eyeball can be moved in no direction except outward (abducens), and outward and downward with rotation of eyeball (superior oblique). For symptoms characteristic of the isolated paralysis of each ocular muscle see Chart XIVc, 818. When the superior oblique muscle is paralysed by an *intra-orbital* lesion the levator palpebrae superioris is usually paralysed with it and ptosis results.	**Paralysis 700 of Motor Oculi. (Figs. 14, 18**
	The ocular muscles, except the levator palpebrae superioris, have a bilateral cortical representation. Hence ocular paralyses, except ptosis, almost never occur in lesions above the oculomotor nucleus, except in bilateral lesions. The cortical representation of the ocular muscles seems to be very diffuse or multiple. Conjugate deviation may result from supra-nuclear lesions.	
	For the symptoms of paralysis of the trochlearis (patheticus) nerve and of the abducens nerve, each of which produces a strabismus convergens, see 818.	**Paralysis 701 of Trochlearis and of Abducens.**
JAW	The muscles of mastication of one side, rarely of both sides, are paralysed and in severe cases atrophied. The temporal and masseter muscles cannot be felt firmly contracting when efforts are made to chew. The jaw cannot be closed tightly or opened strongly or moved laterally towards the healthy side (external pterygoids), or the chin pushed forwards (internal pterygoids). Mastication of food is difficult or impossible; dysmasesis (286). The jaw reflex (323) is abolished. In some cases one side of the soft palate (tensor veli palatini) is paralysed and in some the hearing of low tones is unpleasant (tensor tympani).	**Paralysis 702 of motor branch of Trigeminus.**
	In trigeminus lesions there is unilateral abolition of the conjunctival, corneal, sneezing and palatal reflexes; and the secretion of tears is at times affected. There is no irritation, or tears, from inhaling ammonia or acetic acid. There is also loss of sense of taste, dilation of the pupil, narrowing of the eyelid slit and even enophthalamos. Heat and redness of skin in recent cases and coldness and cyanosis of skin in old cases. The salivary secretion and taste are affected when either the proximal or the distal end, but not the middle, of the nerve is affected.	
FACE	The muscles of expression of one side (facial monoplegia), rarely of both sides, (facial diplegia) of the face are paralysed. The forehead cannot be wrinkled and the eye appears larger than normal and cannot be closed (lagophthalmos, hare's eye). When attempts are made to close the eyelids the eyeball turns upward, the cornea disappearing behind the upper lid (Bell's phenomenon—434). The angle of the mouth is lower than normal and cannot be raised. The naso-labial fold is obliterated. The lips cannot be firmly closed; so that whistling is impossible and speech is impaired. Mastication is difficult, because the weakened buccinator muscle allows food to collect between the jaws and the cheek. The platysma is also paralysed; so that the angle of the mouth cannot be drawn downwards. Tears may flow from the eye and irritate the cheek and saliva from	Facial 703 Paralysis. Bell's palsy. Prosopoplegia. Facial Monoplegia. Facial Diplegia, (751, 928, 1333).

Inability to Move, more or less, Muscles of the	Abstract of Symptoms	Diagnosis
Face (Con.)	the angle of the mouth. The conjunctiva may become inflamed and the cornea ulcerated, because the eyelid cannot wink and keep the conjunctiva clean. In some cases the facial paralysis may be preceded and accompanied by pain. When both sides of the face are paralysed (facial diplegia) the symptoms, just described, are present on both sides. This condition is not quite so easily recognized, because there is no healthy side to compare and contrast with the paralysed one. In severe cases the paralysed muscles exhibit the electrical reaction of degeneration. Hearing and taste are frequently impaired and disordered. When taste is affected the salivary secretion is also affected. In the early stages of the disease the face is drawn over toward the healthy side by the unantagonized healthy muscles. In the later stages the face may be drawn back again permanently towards the paralyzed side by the contracting, newly formed connective tissue in the degenerated muscles. Also in the early stage of recovery the face may be drawn towards the paralyzed side by over-innervation of the muscles formerly paralyzed, and may exhibit temporary contractures and spasms, possibly "associated movements." These spastic symptoms may be due to irregular regeneration of the nerve. The upper fibres of the facial nerve have a bilateral cortical representation as do the laryngeal nerves. Hence lesions of the cerebral hemispheres paralyse mainly the lower branch of the facial; the eye on the paralysed side can be closed, but is easily forced open. For the localization of the different forms of facial paralysis, see 1333.	
Pharynx	The glosso-pharyngeus nerve contains motor as well as sensory fibers and lesions of it, probably, cause partial or complete paralysis of the pharnyx; but no isolated lesion of the glosso-pharyngeus has been recorded.	Glosso-pharyngeus Paralysis 703a
Larynx	Paralysis of the pneumogastric nerve is discussed under 763. In addition to the laryngeal paralysis there are often present disorder of the respiratory act and of the heart beat (tachycardia) and unilateral paralysis of the soft palate.	Pneumo-gastric Paralysis (760). 704
Neck	When the tip of the shoulder sinks downwards and forwards and the arm cannot be easily raised, there may be a paresis of the trapezoid muscle. When this muscle is paralysed on both sides, the head tends to fall forward. When the head is drawn towards one shoulder and the chin turned upwards and towards the other, the sterno-cleido-mastoid muscle is paralysed on that side toward which the chin turns. This posture is called caput obstipum spasticum, when the muscle is atrophied and secondarily contracted and the deformity can no longer be corrected by passive motion. Caput obstipum spasticum occurs also and is more pronounced in torticollis from spasm of the muscle (730). When the sterno-cleido-mastoid muscle is paralysed on both sides, the head tends to fall backwards.	Paralysis of the Spinal Accessory. 705
	When one side of the tongue is paralysed, as frequently happens in hemiplegia, the tongue when protruded turns towards the paralysed side. When both sides are paralysed the tongue can-	

Inability to Move, more or less, Muscles of the	Abstract of Symptoms	Diagnosis	
TONGUE	not be protruded at all, and in such cases, speech, mastication and deglutition are difficult and imperfect. In lesions of the *nucleus* of the hypoglossus nerve there is a paralysis, usually bilateral, associated with patches of muscular atrophy and with tremor. There are often also paralysis and atrophy in groups of muscles in the hands and shoulders (Progressive muscular atrophy—546). There is also a mild paresis of the orbicularis oris muscle. Intracranial lesions involving the hypoglossus and other nerve roots at the base of the brain may cause Avellis' syndrome: pharyngo-laryngeal or glosso-pharyngo-laryngeal paralysis combined with paralysis of the soft palate and, if the hypoglossus nucleus be involved, of one-half of the tongue also; or may cause Schmidt's syndrome: the above and also sterno-cleido-mastoid and trapezius paralysis. In lesions of the cortical hypoglossus area, even unilateral ones, a bilateral paralysis of the tongue may result, without atrophy and without any change in its electrical reactions.	Hypoglossus Paralysis. (546, 755).	70
DIAPHRAGM	The diaphragm is paralysed on one or both sides, causing dyspnoea on exertion and sinking in of the epigastrium on inspiration, especially on deep inspiration. The lower part of the lung is drawn upwards and atolectasis and pneumonia may occur. Besides the usual causes of compression and neuritis, this paralysis may also occur in pleurisy, peritonitis, trichinosis and in bulbar and spinal lesions. The paralysed diaphragm shows Litten's phenomenon.	Phrenic Paralysis.	70
ARM	The supra and infra-spinatus muscles are paralysed; so that rotation of the arm outward and raising it in abduction are impaired. Muscles involved are atrophic and ulnar side of hand is turned forwards.	Supra-Scapular Paralysis.	70
	The serratus anticus major is paralysed: so that when the scapula is raised, its lower angle approaches the vertebrae and the inner margin of the scapula does not lie close to the thorax and, on movements of the arm upwards and forwards, stands from the thorax like a wing. The arm cannot be raised beyond a horizontal line.	Long Thoracic Paralysis. Serratus Paralysis.	70
	Motion of the arm inward and forward is impaired. Hand cannot be placed on opposite shoulder.	Anterior and Posterior Thoracic Paralysis.	71
	Rotation of the arm inward and motion of the arm backward are impaired.	Sub-Scapular Paralysis.	71
	The deltoid and teres minor are paralysed: so that the arm cannot be raised.	Axillary Paralysis.	71
	The combined paralyses of the brachial plexus: Erb's and Klumpke's paralysis, are discussed under 444, 445 and 490.		
	The biceps, brachialis anticus and coraco-brachialis muscles are more or less completely paralysed; so that flexion of the arm at elbow is more or less impaired, especially in supination (very rare.)	Musculo-Cutaneous Paralysis.	71

Inability to Move, more or Less, Muscles of the	Abstract of Symptoms	Diagnosis
HAND	The pronators and flexors of the hands and fingers, the muscles of the ball of the thumb and the first and second lumbrical muscles are paralysed. The hand can neither be flexed nor pronated. The thumb cannot be brought across the hand to touch the little finger, but remains close to the index finger (ape's hand). The first (proximal) phalanges of fingers can be flexed, but not the second and third phalanges.	Median Paralysis. 714
	The interossei, the third and fourth lumbricales, and the muscles of the little finger are paralysed. The proximal phalanges cannot be flexed, the other phalanges cannot be extended and the little finger cannot be moved. The fingers cannot be spread. When muscle atrophy and contracture occur "claw hand" results.	Ulnar Paralysis. 715
	The extensors and supinators of the hand and fingers and the abductor pollicis longus are paralysed. The thumb is adducted and can neither be abducted nor extended. Wrist-drop and slight pronation. Wrist and fingers cannot be extended completely. The wrist-drop differs from that of lead palsy (494) in that the supinator longus is paralysed. Therefore, if the forearm is held midway between supination and pronation and the elbow strongly flexed against a resistance offered, the belly of the supinator longus will not stand out firmly contracted as it will in lead paralysis and in health.	Musculo-Spiral and Radial Paralysis. 716
LEG	The extensor femoris is paralysed; so that flexion of the thigh on the body and extension of leg on thigh are impossible or difficult. Standing and walking are difficult, and ascension, jumping and running impossible. Knee-jerk usually absent.	Crural Paralysis (997). 717
	The adductor muscles of thigh are paralysed; so that adduction of leg, pressing of thighs together and crossing of legs are impossible.	Obturator Paralysis. 718
	The glutei muscles are paralysed; so that walking, ascending stairs, straightening up of body, abduction and rotation of thigh are impaired. Generally much muscular atrophy.	Gluteal Paralysis. 719
	Foot and toes are paralysed; the leg cannot be flexed at knee and rotation of the thigh is impaired. In cases of isolated tibialis paralysis there is absence of plantar flexion of foot, and of plantar flexion, spreading and adduction of toes (Pes calcaneus et valgus.) In cases of isolated peroneal paralysis there is absence of dorsal flexion and abduction of foot and its adduction impaired—absence of dorsal flexion of toes. There are foot-drop, high stepping gait and Pes equino-varus.	Sciatic, Tibial and Peroneal Paralysis. (996). 720
	For paralysis from lesions of the cauda-equina, see 487, 1007 and 1308.	Cauda Equina Paralysis. (Fig. 29). 721

CHART XIId

Local Spasms

Comprising Numbers 725 to 733 on right margin.

637—LOCAL SPASMS

Spasm of Muscles of	Abstract of Symptoms	Diagnosis	
EYE	For spasm of ocular muscles, see XIV, 878.		
JAW	The jaws are held tightly shut and the masseter and temporal muscles can be felt to be contracted (lock jaw), usually bilaterally. The spasm may be "tonic," as in tetanus (603), tetany (612), irritation of teeth (wisdom teeth) and certain unilateral lesions of the pons and medulla; or "clonic," as in chills and in rare cases of paralysis agitans and hysteria. When the pterygoid muscles alone are in spasm the mouth is held open and cannot be closed.	Trigeminal Spasm or Cramp. Trismus.	725
FACE	Spasms of one or more muscles of expression of the face, unilateral or bilateral, are relatively common, as in convulsive tic (598) and tic douloureux (599). These spasms are often a mixture of tonic and clonic contractions, the clonic predominating. They may affect all the muscles or only one, as in tonic spasm of the orbicularis palpebrarum (blepharospasm) (598, 616), or in clonic spasm of this muscle (spasmus nictitans: nictitation). The platysma myoides often takes part in these spasms and very rarely the muscles of the soft palate and the internal and external ear muscles. Very rarely spasm of some of the facial muscles about the mouth constitutes an occupation neurosis or cramp, as in the "Auctioneer's cramp" and "Cornet player's cramp." These facial cramps may be symptomatic directly of lesions of the cortical facial center, of the facial nerve in its course, and reflexly of the trigeminal nerve or its terminal filaments in the eye, nose, mouth or ear. There are also to be remembered the passive contracture of the degenerated muscles and the active contracture due to over-innervation of the convalescing muscles in facial paralysis. Causeless and uncontrollable laughter must also be classed among the facial spasms. This condition, similar to the allied state of causeless and uncontrollable crying, occurs especially in hysteria and in lesions of the optic thalamus.	Facial Spasm or Cramp (267, 598-9).	726
PHARYNX	Spasm of the pharynx of a tonic nature preventing swallowing and of a clonic nature repeating the act of swallowing with great frequency occur. The former occurs in hydrophobia (604) and somewhat also in tetanus (603); while the latter, associated with coma, frequently occurs in mild epileptic attacks. The spasm also occurs from irritation of the pharynx in hysteria and very rarely, as one of the crises in locomotor ataxia (423). Spasm of the oesphagus is not uncommon in hysterical persons and makes the swallowing of food very difficult.	Glossopharyngeal Spasm or Cramp.	727
LARYNX	Spasm of the muscles of the larynx (spasmus glottidis, false croup, laryngismus stridulus), causing noisy and difficult breathing, is a not uncommon and occasionally a dangerous condition. It occurs almost exclusively in children and is often associated with rickets and with digestive disorders. Occurs also in general diseases such as hydrophobia, hysteria, epilepsy, chorea, tabetic crises, etc. Sneezing (sternutatio spastica, ptarmus) and coughing, reflex acts implicating both the pneumogastric and the intercostal nerves, are often due to pathological conditions and irritation of the nervous system. Bradycardia, Cheyne-Stokes' respiration and cerebral vomiting are symptoms of irritation of the pneumogastric nucleus, but are not characteristic and are of little diagnostic value.	Pneumogastric Spasm or Cramp.	728

Spasm of Muscles of	Abstract of Symptoms	Diagnosis
TONGUE	Spasm of the tongue is very rare, especially so the tonic form. During the attack speaking and swallowing are impossible. Very rarely a tonic spasm of the tongue occurs when the patient attempts to speak (stuttering and aphthongia). Spasm of the tongue is sometimes associated with facial spasm and with spasm fo the submaxillary muscles. These spasms may be due directly to lesions of the cortical tongue center, of the hypoglossus nerve in its course, or, reflexly, especially from lesions of teeth, mouth and nose.	Hypoglossus Spasm or Cramp. (770-1). 729
NECK	Spasm of the neck muscles, especially the sterno-cleido-mastoid, caput obstipum (spastic wry neck), is sometimes congenital and is sometimes acquired in later life. In these cases the head is drawn toward the shoulder of the affected side and the chin is turned toward the other side and slightly elevated and the sterno-cleido-mastoid muscle can be felt to be firmly contracted. When the trapezius is the seat of the spasm the occiput is drawn backwards and turned toward the shoulder of the affected side and the edge of the muscle can be felt to be firmly contracted. Spasm of the muscles is sometimes tonic, sometimes clonic and often both. The cause of these spasms is often neurotic and often rheumatic. Rarely it is some disease of the eye or of the ear (torticollis ab oculo laeso, ab aure laesa) or of the cervical vertebrae. Usually many muscles are involved, although one or two more prominently than the others. Spasm of the neck muscles with retraction of the head is a prominent symptom in meningitis and strychnine poisoning.	Spinal Accessory Spasm or Cramp (598). 730
DIAPHRAGM	Tonic spasm of the diaphragm, either unilateral or bilateral, occurs very rarely and produces dangerous dyspnoea. It sometimes occurs as one symptom of a general disease: tetanus, hydrophobia, hysteria, etc. Clonic contractions are common and cause hiccough (singultus), always a distressing and at times a dangerous symptom, which occurs occasionally in brain and spinal cord lesions and frequently in irritation of the pneumogastric nerve, especially from the gastric mucous membrane. A similar but slower contraction of the diaphragm associated with facial spasm (opening of mouth) causes the act of yawning (oscedo, chasmus) which is sometimes frequently repeated as an aura of apoplexy or epilepsy and occurs also in hysteria, digestive disorders, drowsiness, etc.	Phrenic Spasm or Cramp. 731
ABDOMEN	Tonic and clonic contractions of some or all of the abdominal muscles occur with extreme rarity, and the latter are usually, if not always, hysterical. Rigidity and retraction of the abdomen occur in meningitis, peritonitis and in perforation of stomach or bowels. Local or general rigidity occurs in appendicitis, rupture of the Fallopian tube and the varous colics.	Intercostal Spasm. Abdominal Spasm. 732
ARM & LEG	Tonic and clonic spasms of the muscles of the arm and shoulder or of the leg, with the exception of the secondary contractures due to lesions of the pyramidal tract and of the peripheral nerves, are very rare. They usually are due either to deficiency of water in the system, and often occur in disease in which much water is lost, as cholera, diarrhoea, etc., or to hysteria, or to rheumatic factors, or are reflex. The deformity resulting in each case can be predicted from the function of the muscle involved.	Brachial, or Lumbar, or Sciatic Plexus, Spasm or Cramp. 733

CHART XIII

Disorders of Speech and Gait

DIAGNOSITIC ANALYSIS OF SYMPTOMS

Symptoms Analysed	Character of Disorder	
735 DISORDERS OF SPEECH, READING AND WRITING.	737 ANARTHRIA (283) Inability or unwillingness to speak. No disease of vocal organs or peripheral nerves. This condition may result from a complete aphonia (260) or complete aphasia (222) or complete dysarthria (284), or delusions (1112), or dementia (1095), or voluntarily.	The diseases in which Anarthria and Dysarthria occur are set forth in Chart XIIIa.
	738 DYSARTHRIA (284) Ability to express thought by speech but articulation is defective.	
	739 APHASIA (222 to 233) Articulation normal but expression of normal thought is defective.	The varieties of Aphasia and the conditions under which they occur are set forth in Chart XIIIb.
736 DISORDERS OF GAIT.	740 ATAXIC	The diseases in which Disorders of Gait occur are set forth in Chart XIIIc.
	741 PARALYTIC AND FLACCID	
	742 PARALYTIC AND SPASTIC	

CHART XIIIa

Anarthria and Dysarthria

Comprising Numbers 737 and 738 on left side of Chart
and 743 to 771 on right margin

DIAGNOSTIC SYMPTOMS AND TESTS

737 ANARTHRIA (283)

- **Result of disease in infancy, or congenital.**
  - Auditory memories necessary for understanding spoken words were never acquired, or early lost through disease; hence innervation memories necessary for speech were never learned.
    - May make noise but cannot
    - Can be trained to speak thr sight.
- **Result of disease in adult life.**
  - Innervation memories necessary for speech have been acquired but are not available. No hysterical symptoms.
    - Complete absence of speech, is impossible. Patient m speak or to communicate by
  - Hysterical symptoms and etiological factors present, although not always prominent.
    - Will neither whisper nor spe
    - Can whisper faintly but disti
  - Apoplectic symptoms (504).
    - May mutter but cannot artic
    - Loss of speech may be comple

738 DYSARTHRIA (284, 1401)

- **Congenital.**
  - Vocal organs defective.
    - Words imperfectly formed, also a nasal vo
  - Vocal organs normal.
    - Words imperfectly formed and usually a v
- **Defective Education.**
  - Vocal organs normal.
    - Substitution of one letter for another. An speaks the vowels correctly but has diffi
- **Paralytic.**
  - The labials, the linguals or the vowel sounds or all of them cannot be properly pronounced. A careful examination reveals a paralysis or a paresis within the domain of the facial, the hypoglossal or the pneumogastic nerve.
    - No symtoms of any central disease.
      - Patient cannot whistle or close lips tightly.
      - Tongue is not protruded straight but devia
      - Soft palate is not raised (bilateral) or no
      - Anesthesia of larynx. Paralysis of crico- and on lower level) and of thyreo-ary-e
      - Immobility of one or both vocal cords from veric position of cords (between extrem in cases of unilateral paralysis, the hea paralysed cord.
      - Immobility of one or both vocal cords fro cord or cords lie near the median line become smaller on inspiration.
      - Immobility of one or both vocal cords f arytenoid lateralis muscles) and in som weak and hoarse. Cords are wide open concave.
    - May be symptoms of central disease.
      - Unilateral or bilateral paralysis of t of all the laryngeal muscles and larynx.
- **Tremor and Ataxia.**
  - Slow and clumsy speech.
    - Cerebellar gait.
    - Speech sounds as if
  - Tremulous and slovenly speech, words are badly formed, letters and syllables are left out both in speaking and writing.
    - Evident mental deterioration.
      - Argyll-Robertson's attacks may occu
      - Alcoholic history, a
  - Scanning speech.
    - Intention Tremor.
    - Great variety of w
- **Rigidity.**
  - Monotonous speech.
    - Passive Tremor.
    - Rigidity of muscles
- **Spasm.**
  - Certain letters (consonants) are spoken with difficulty and are repeated many times
  - Utterance is arrested by a spasm of one or more of the muscles concerned in spee is directed to the speech the worse it becomes. Singing is usually not at all aff

CHART XIIIb
Amnesia and Aphasia

Comprising Numbers 739 on left side of Chart
and 772 to 780 on right margin

	TEST	ABSTRACT
739 AMNESIA AND APHASIA (220 to 229). None of these conditions constitutes a disease, but is rather one symptom of a more complex disease. Each is a form of dementia, or more accurately, amnesia in the broad sense of the term and consists in a loss of general or special memories. See also Anarthria and Dysarthria (737-8). In examining patients for amnesia or aphasia, it is most important to first ascertain to what degree, if at all, they are deaf.	Patient is capable of normal speech but exhibits a decided loss of memory.	The loss of memory may not be accompanied by any, or the names of persons is rather common and of no di less distant past, are referred by the memory to the cerebral concussion and compression (1042-3), espec little time immediately previous to the injury and fr Examination of the patient shows a loss of memory, es in old people and in the insane, and is usually associ
	Patient is incapable of normal speech (spontaneous, repeating after dictation, reading aloud) for want of innervation memories of a few or many spoken words.	Can express ideas by gestures, but cannot name objects verbs better than nouns and proper names. Recogn tient is frequently at a loss for a word. His vocabul phasia—778) but is often conscious of his mistake if matismus), but can often repeat sequences of num from copy, but makes many mistakes in spontaneous with right-sided hemiplegia in right-handed persons a
	Patient is incapable of normal speech for want of auditory memories of spoken words.	Patient fails to understand more or less of what is sai what he repeats. Cannot execute verbal commands, conscious of this mistake even when his attention is
	Patient is incapable of normal speech for want of visual memories of objects.	Patient cannot name objects seen, but may, at times, aphasia has been classed under the complex term "m
	Patient is incapable of normal speech for want of visual memories of written or printed words.	Patient cannot read written or printed letters or word use a wrong word and, when they do so, are conscio many mistakes in spontaneous writing. Patients ca
	Patient is incapable of normal speech, because of a faulty co-operation of the various cortical centers concerned in speech.	Patients suffering from this defect exhibit a combinatio groups. First, those in which the symptoms of moto combination, incomplete, of the symptoms of motor group (Transcortical motor aphasia) spontaneous of the year, the alphabet, numerals, etc.) are little, group (Transcortical sensory aphasia) there is more (days, months, etc.,) are possible; although these w gree of both sensory and motor aphasia. Spontaneo three groups resemble those occurring in gradual rec impossible or defective. The patient omits words in so that the speech or writing, when possible at all.
AGRAPHIA.	Patient's speech is normal, but his writing is abnormal.	Patient is incapable of spontaneous writing for want simply cannot write. A very rare condition un left frontal convolution is immediately above the mo printed matter into script. His copying is purely m Patient omits words in writing, uses the wrong words,

The subject of aphasia, in its various forms and subdivisions, is a very complicated one and is, as yet, far from being solved. Too f

Broca, in 1861, published a case of motor aphasia with a lesion at the base of the left inferior frontal convolution and thereby laid

Wernicke (whose studies have contributed greatly to the comprehension of aphasia) divided motor and sensory aphasia into three s

1st. Cortical Motor Aphasia, in which the patient is unable to speak, write or read aloud correctly, or to speak or write correct

2nd. Subcortical Motor Aphasia, in which the patient can neither speak spontaneously nor from dictation nor read aloud correc

3rd. Transcortical Motor Aphasia, in which the patient can neither speak nor write correctly, but can speak and write from di

1st. Cortical Sensory Aphasia, in which the patient can speak (with paraphasia) and copy, but can neither write, nor speak,

2nd. Sub-cortical Sensory Aphasia, in which the patient can speak quite perfectly, write, copy, read aloud and understand wr

3rd. Transcortical Sensory Aphasia, in which the patient can speak (with paraphasia) and write (with paragraphia), can cop

Wernicke also recognizes a Conduction Aphasia, in which the patient can speak, write and read and understand correctly, but exhi

Marie considers all forms of aphasia as resulting from a greater or less degree of a general intellectual impairment rather than from aphasia with difficulty of articulation (anarthria or dysarthria). Whether he is altogether right in this or not, certainly our conce nicke's ideas, neither of which is probably altogether false. It is to be remembered, however, that many cases of dementia, in all stages acteristic, defects of one or more of the component parts of the complex phenomenon of speech. This strongly indicates a loss of so tion is, at the present day, too firmly proved to be easily abandoned.

OF SYMPTOMS

ND AGRAPHIA

YMPTOMS	DIAGNOSIS	
y very little, intellectual impairment in other respects. To a certain degree the loss of memory of ic or prognostic value. "Retroactive amnesia" is where events, which occurred in the more or iate past, as in Korsakoff's psychosis (1102). "Retrograde amnesia" occurs in some cases of hose associated with fright. In it, memory is lost of those events which occurred during some	Amnesia.	772
y for recent events, impaired judgment and a general failure of mental powers. Very common ith mental depression.	Dementia (1079).	773
or at all. Patient knows the idea he wishes to express, but cannot put it into words. Can use e desired word when it is spoken to him and can often then pronounce it. In speaking, the pa- limited often to one or two words, or even to none (anarthria—737). Uses a wrong word (para- tention is called to it and often when it is not. Cannot construct sentences correctly (agram- ays, months, etc., and can sing songs. When his arm is not paralysed patient can usually write g (paragraphia—780). Can usually read but not aloud. The condition is usually associated -versa.	Motor Aphasia or Aphemia (222, 1402).	774
m. Cannot repeat what is said to him, or if in rare cases he can do this, he does not understand dily executes written ones. In speaking, the patient frequently uses a wrong word and is not o it. Can write spontaneously and from copy but not from dictation. He can read well.	Sensory Aphasia. Auditory Aphasia. Word Deafness (222-33, 1382-82a).	775
ze and name objects which he touches and feels. His defect in speech is not great. Visual hasia," of which it may form a not inconsiderable part.	Visual or Optic Aphasia (224, 232, 1357).	776
cannot execute written commands, but readily executes verbal ones. In speaking, patients rarely eir mistakes. Patient can write from dictation imperfectly, but not at all from copy and makes d what they have written.	Alexia. Word Blindness (229, 1382a, 1403).	777
tor (774) and sensory (775-6-7) aphasia in varying degree and can be classified into three ia predominate. Second, those in which the symptoms of sensory aphasia predominate. Third, a sory aphasia. A sharp line, however, cannot be drawn between these three groups. In the first is disturbed; but repeating after dictation and reciting of serials (days of the week, the months l, impaired. Cases in this group run rather a rapid course towards recovery. In the second fully developed sensory aphasia and the repeating after dictation and the reciting of serials not understood when spoken by themselves or others. In the third group there is some de- ch is very imperfect; while the reciting of serials is well preserved. The symptoms in all rom pure motor or sensory aphasia. In all three groups reading aloud and writing are either g or writing, or uses a wrong word (paraphasia), or puts a right word in a wrong place; comes quite incoherent (Jargon speech).	Transcortical Aphasia. Mixed Aphasia (225). Paraphasia (226).	778
necessary innervation memories. His arm and hand are not paralyzed for other movements. He ated by motor aphasia, because the cortical area for writing being at the base of the second ch center. A patient with agraphia may be able to copy print or script, but he cannot copy al. A patient may be able to write letters, spontaneously, but not words and sentences.	Agraphia (227, 1389).	779
p words in the sentences so that writing becomes incoherent. He can, however, write.	Paragraphia (226).	780

s, scientifically observed, have come to autopsy.

undation, not only of the modern ideas about the faculty of speech, but also of cerebral localization.

ions each:

dictation, or to read with full understanding, but can copy correctly and understands what is said to him.

can read, write and understand what is said to him.

can copy, can read aloud, and can understand speech and writing.

from dictation, nor read aloud perfectly, nor understand speech or writing.

cannot speak or write from dictation, nor understand speech.

nd speak from dictation, and read aloud but all without understanding, and cannot understand either speech or writing.

phasia and paragraphia.

bral lesions, especially not to those of the left inferior frontal convolution. He considers motor aphasia to be a combination of sensory aphasia previously to Marie's article had been growing too schematic. The truth probably lies somewhere between Marie's and Wer- pment, exhibit no aphasia. Furthermore, cases of aphasia are rarely permanently complete (anarthria), but show varying, even char- t of a composite whole and each of these elements may, in time, be more certainly localized. The theory of a cerebral cortical localiza-

CHART XIIIc

Disorders of Gait; Ataxic, Paralytic and Flaccid, Paralytic and Spastic Gaits

Comprising Numbers 736 to 742 on left side of Chart
and 781 to 804 on right margin

(Note)—In addition to the diseases mentioned in this chart, pain, whether in the joints (rheumatism, gout, arthritis, morbus coxae, etc.), or in the muscles (rheumatism myositis etc.), or in the bones (caries, etc.), or in the nerves (sciatica, etc.) will cause a limping gait. The gait of a patient suffering from weakened arches in the feet is very characteristic in advanced cases and this common disease should always be thought of in any disturbance of gait and in any painful affection of the legs or lower back.

- **736 DISORDERS OF GAIT.**
  - **740 Ataxia. (Incoordination is the most prominent symptom.) (See also 799.)**
    - Staggering Gait. (Reeling gait.)
      - The disorder is of a **temporary nature.** Patient's speech is blurred and foolish.
      - Disease of permanent nature. Patient sways from side to side and lurches like a drunken man. The ataxia is almost entirely limited to walking and standing.
        - There is a strong heredity and disease occurs in family groups and in youth. Nystagmus.
        - No heredity. Occurs at any age.
    - Incoordinated Gait. (Stamping Gait.)
      - Patient does not walk like a drunken man, but throws his legs about in an aw and excessive manner. All movements of legs are ataxic. In well marked case are raised high, flung outwards and forwards excessively and brought back do ground with hard stamp on heel. The eyes are employed to control the move and walking in the dark is very imperfect or impossible.
  - **741 Paralytic and flaccid. (Weakness is the most prominent symptom.)**
    - Waddling Gait.
      - Muscular atrophy and pseudo-hypertrophy.
        - In walking patient throws body from of others, but all are weak. In risin seen at times in pregnancy and in a
        - Muscles normal.
        - Similar walk. Congenital. Usually b shows dislocation of hip and absence
    - High-stepping Gait.
      - General weakness, especially of extensors. Bilateral. May be some ataxia in t symptoms.
      - Weakness of extensors only. Bilateral. Blue line on gums. Wrist-drop as well
      - Variable distribution. Weakness, especially of extensors. Often unilateral. M
    - Feet drag over ground.
      - In walking all muscles of legs seem too weak to raise feet. No tremor or spasm. Steps short.
        - Temporary condition following illness.
        - Permanent condition. Organic and pe able to walk in this disease.
        - Advanced age, atheromatous arteries.
    - Inability to stand on one or both feet.
      - Hysterical symptoms present. Lack of will power while pretending to have much. Knee-jerk may be increased. Faint ankle-clonus often, Babinski always, absent.
        - Both legs. — Legs can be moved freely and normal parently is afraid to walk or has for quent, legs give way under her and
        - One leg. — The weak leg is drawn along after the shows more strength in leg than wou moves badly in each direction (Schi
        - Unilateral. — The weak leg is usually swung forwar (mowing gait). The leg is usually whole. In walking sideways (stepp wards the healthy side (Schüller's si
        - Bilateral.
      - Tendon reflexes increased. Ankle-clonus and Babinski present.
  - **742 Paralytic and spastic (Stiffness is the most prominent symptom.)**
    - Toes scrape along ground. Legs rigid and frequently tremble.
      - The legs are rigid and offer resistance so that, in walking, body and shoulde backwards to pull legs forwards. trembling (clonus) when brought f ducted so that knees are held tight in walking (scissors gait). Pernic of severe, anemia may be present.
      - General rigidity. — Patient is slightly bent forwards and all his joints slight retropulsion—a tendency to stagger backwards. Pass

	DIAGNOSIS	
isorder and history of alcoholic abuse.	Alcoholic Intoxication (658, 663, 673, 767).	781
erty. Knee-jerks usually absent. Contracture and deformity of feet. Babinski	Friedreich's or Hereditary Ataxia (651, 670, 687, 765).	782
erty but in youth. Knee-jerks usually present and exaggerated. Oculo-motor optic atrophy.	Marie's or Hereditary, Cerebellar Ataxia (650, 669).	783
d, cerebellar fits and other cerebellar symptoms may be present.	Lesions of Cerebellum or its tracts (607-8-47, 693, 1016, 1295).	784
rks abolished. Argyll-Robertson's phenomenon, optic atrophy. History of lis usually. A common disease.	Tabes (661, 759, 827, 896, 979, 988, 1004, 1217, 1231). (Figs. 24-7).	785
rks are usually present. May be no other symptoms than ataxia and anes-, or may be all the spinal symptoms of locomotor ataxia, but none of the cra-especially no eye symptoms. A rare disease.	Lesions of posterior columns of spinal cord (654, 1271, 1360-3-4, 1406). (Figs. 24-6).	786
a duck. Marked lordosis. Atrophy of some muscles, apparent hypertrophy himself up with his hands and crawls up upon his own legs. (A similar gait is	Muscular Dystrophies (477, 1154).	787
be unilateral. No change in the muscles. Hip joints unusualy mobile. X-ray	Congenital Dislocation of the Hip.	788
lar weakness, tenderness and atrophy. Knee-jerks absent. Many sensory	Multiple Neuritis (488, 662, 825, 1008, 1147, 1327).	789
History of colic and of exposure to lead.	Lead Palsy (494, 584, 1053).	790
without tenderness. Electrical reaction of degenreation. No sensory symptoms.	Acute Anterior Poliomyelitis (495, 1148, 1233). (Figs. 24-7).	791
peripheral reflexes normal. No sensory paralysis.	Weakness (671).	792
disordered (lost). Sensory paralysis. Patients, even with crutches, are rarely	Myelitis or Myelomalacia in lumbar enlargement of cord (485, 827, 1149, 1329).	793
r and mental impairment. Reflexes normal or increased.	Senile Paraplegia (522).	794
: sitting. Patient apparently makes no effort to walk. Legs collapse. Ap-alk. In other cases, patient walks normally; but at intervals, usually fre-ver hurts herself seriously.	Astasia and Abasia (287, 652).	795
never advances beyond it. In some actions, when taken unawares, the patient for walking. In walking sideways (stepping laterally) along a line patient 456).	Hysterical Hemiplegia or Monoplegia (527, 1076).	796
about the normal leg as a pivot and is set down in advance of this latter l at the knee and the whole side of the body is rigid and swings forward as a ng a line the patient moves well towards the paralysed side, but badly to-	Organic Hemiplegia or Monoplegia. (Apoplexy, Cerebral or Spinal Tumor or Abscess).	797
Organic reflexes are disordered, and sensory symptoms are present. No ataxia.	Myelitis or Myelomalacia above lumbar enlargement, including Compression Myelitis (513-4-7-20-50, 831). (Figs. 24-7).	798
Organic reflexes may or may not be disordered, sensory symptoms. Marked ataxia.	Ataxic Paraplegia (526, 660). (Figs. 24-7).	799
Organic reflexes not disordered. No sensory symptoms. No ataxia. — Adult.	Spastic Paraplegia (525), including Amyotrophic Lateral Sclerosis (547).	800
Organic reflexes not disordered. No sensory symptoms. No ataxia. — Youth. Scissors Gait.	Cerebral Diplegia (478, 501, 577, 1051).	801
Dissociation of sensation (365).	Syringomyelia (553, 698, 840-2, 1009, 1152, 1170, 1187, 1370-2).	802
Intention tremor, marked ataxia, at times staggering gait.	Disseminated or Multiple Sclerosis (511, 580, 659, 666, 688, 759, 768, 914, 1054).	803
tion and propulsion—a tendency to go forward at ever increasing speed; also also Encephalitis Lethargica (1047).	Paralysis Agitans (610, 677, 769).	804

ements; bent far y show are ad- crossed or form

CHART XIV

Disorders of Sensation

DIAGNOSTIC ANALYSIS OF SYMPTOMS

DISORDERS OF GENERAL SENSATION AND OF THE SPECIAL SENSES

Symptom Analysed	Alterations in Sensation		
805 Disorders of Sensation. (See also Perversion of Sensation Chart XV).	806 Diminution of Sensation.	811 Anesthesia and Analgesia.	See Chart XIV a.
		812 Dissociation of Sensation.	
		813 Loss of Muscle Sense.	
		814 Numbness.	
	807 Exaggeration of Sensation.	815 Hyperesthesia.	
	808 Disturbances of Vision.	816 Perversion.	See Chart XIV b.
		817 Limitation of field of vision.	
		818 Double vision (Diplopia).	See Chart XIV c.
		819 Conjugate Deviation of Eyeballs.	
		820 Pupillary Abnormalities.	See Chart XIV d.
		821 Ophthalmoscopic Examination.	
	809 Disturbances of Hearing.	822 Deafness (anakusia).	See Chart XIV e.
	810 Disturbances of Taste and Smell.	823 Hyperakusia (oxyakoia) or Parakusia.	

CHART XIVa

Disorders of Sensation

Comprising Numbers 806 to 814 on left side of Chart
and 824 to 844 on right margin

CHART XIVb

Disturbances of Vision

Comprising Numbers 808, 816, 817 on left side of Chart
and 845 to 871 on right margin

Blindness may be caused by a solitary lesion in the eye, or optic nerve, or optic chiasm Hemianopia may be due to a lesion of the optic tract, or geniculate bodies, or fasiculus of Gratiolet, or of the carcarine fissure region on the median surface of the occipital lobe. The cortical center of sight is the cortex of the occipital lobe. The fibers of the optic nerve having their origin in the lower left quadrant of the retinal of both eyes finally terminate in the lower margin of the calcarine fissures of the left occipital lobe (Fige. 16 and 37), and the fibers from the upper left quadrant of the retinal of both eyes terminate in the upper margin of the calcarine fissure of the left occipital lobe; and the same relationship exists between the fibers from the right side of the retinal of both eyes and the calcerine area of the right occipital lobe. The central fibers of the optic nerve having their origin from the small area of clear vision may possibly terminate in, or near, the geniculate bodies, but more probably pass in the fasiculus of Gratiolet to the occipital lobe and terminate either in the floor of the calcerine fissure or more widely in the occipital cortex.

DIAGNOSTIC SYMPTOMS

- 808 DISTURBANCES OF VISION.
  - 816 PERVERSION.
    - A yellow color of all objects seen irrespective of their true color: xanth
    - A red color (erythropsia) of all objects seen irrespective of their true col
    - A green color of all objects seen irrespective of their true color (green
    - Muscae volitantes, twisted threads and irregular spots moving about in
    - Flashes of light and dark spots surrounded by a bright zone (glittering
    - Achromatopsia (364) and hemichromatopsia occur in slight lesions of the
    - An inversion (red having a larger field than the blue—14) and an interlacing of the color fields (Dyschromatopsia).
      - Hysterical symptoms (415)
      - Choked disc and other sympt creased intra-cranial press
  - 817 ABSENCE OR LIMITATION OF FIELD OF VISION (358 to 362-3).
    - BLINDNESS (358, 1334).
      - Peripheral cause.
        - Ocular lesions, such as cata
        - Quinine, in toxic doses, may dilatation of the pupil. I
      - Central cause.
        - Bilateral. No lesion in eye. Pupillary
        - Unilateral
          - No lesion in eye. Optic ne
          - No lesion in eye. No optic may be shown that the bli
    - Homonymous Tetartanopia or Quadrant Hemianopia. No hemiopic pupillary reflex or other paralysis. May disc. Very rarely occurs i or optic fasciculus of oppo
    - Homonymous hemianopia (14, 362, 1337) may very rarely be bilateral, due to double lesion.
      - No hemianesthesia. No he (26)
      - Hemianesthesia.
      - May or may not be a h pupillary reflex. Paralys abducens nerve or both.
    - Bitemporal hemianopia (362, 1335).
    - Nasal hemianopia (362, 1336).
      - Slow onset, progressive cours in complete blindness. Cl pupillary reflex.
    - Horizontal hemianopia. Occurs in lesions of the ret
    - Homonymous scotomata. These may occur as the res neighborhood of the calca
    - Concentric limitation of field of vision, even to complete blindness.
      - Increased tension of eyeball.
      - No increased tension of eyeball.
      - Hysterical symptoms (415)
      - Symptoms of tabes are pre or no ataxia. History of

S OF SYMPTOMS

ɴF VISION

ʔMPTOMS	DIAGNOSIS	
sion).	Jaundice. or Santonin. Amyl Nitrite. Cannabis Indica or Picric Acid Poisoning.	845
	Neurasthenia. Hysteria, great emotional excitement and after cataract operations; also after the eye has been exposed for a long time to an electric or other bright light (snow-blindness).	846
	Diseases of optic nerve and retina and after cataract operations.	847
Seen especially when eyes are turned towards a bright light.	Neurasthenia, circulatory disturbances in brain and digestive disturbances.	848
denly appearing and disappearing in the field of vision.	Migraine and Aura of Epilepsy and circulatory disturbances in brain (575, 858, 1028-61).	849
s. of the optic fasciculus and especially of the calcarine cortex.	Achromatopsia (364).	850
	Hysteria (1076).	851
ease are present. The color field becomes normal after the in- (Cushing.)	Cerebral Tumor (836).	852
tis, retinitis, glaucoma, etc., may cause blindness.	Ocular lesions.	852a
pasm of the retinal arteries. Belladonna may cause extreme ndness, or semi-blindness, may result.	Drugs.	852b
l. Uremic amaurosis may be in this class (edema).	Lesion or edema of both occipital lobes.	853
esent. Pupillary reflexes absent.	Lesion of optic nerve or chiasm (894).	854
ary reflexes normal. Hysterical symptoms. By tests (12) it al.	Hysterical Amblyopia.	855
thesia hoked tract — Upper homonymous quadrant of each field of vision.	Lesion of lower lip of contralateral calcarine fissure.	856
tract — Lower homonymous quadrant of each field of vision.	Lesion of upper lip of contralateral calcarine fissure.	857
reflex ılysis. — Sudden onset and of short duration. Often more marked in, or limited to, one eye. No other symptoms except nervousness. Circulatory disturbances.	Aura of migraine.	858
reflex ılysis. — Choked disc. Slow onset. Progressive course of the disease.	Tumor involving median surface of contralateral occipital lobe or fasciculus of Gratiolet (1378).	859
reflex ılysis. — No choked disc. Rapid onset. Permanent, not progressive, or rarely shows a regressive course.	Hemorrhage or softening in or near contralateral calcanine fissure or optic fasciculus of Gratiolet (1378).	860
No hemiopic pupillary reflex. No choked disc. Regressive course.	Hemorrhage or softening in the posterior part of posterior limb of contralateral internal capsule.	861
ıiopic li or — Choked disc. Slow onset. Progressive course.	Tumor involving contralateral optic tract or geniculate bodies (895, 1337).	862
ıiopic li or — No choked disc. Rapid onset. Symptoms of meningitis may be present.	Neuritis or lesion of contralateral optic tract (895, 1337)	863
ually iopic — Bilateral.	Tumor compressing central part of optic chiasm (894, 1335). (Enlarged pituitary).	864
ually iopic — Unilateral.	Tumor compressing homolateral outer part of optic chiasm (894, 1336).	865
erve or chiasm, invloving their upper or lower portion.	Horizontal hemianopia.	866
ns in the geniculate bodies, in the optic fasciculus or in the	Homonymous scotomata	867
d final atrophy of optic nerve. Pupils dilated and unequal. disc.	Glaucoma (944).	868
scopic examination the optic papilla shows atrophy.	Optic atrophy (898).	869
	Hysteria (1076).	870
gyll-Robertson phenomenon and absence of knee-jerk. Little in and lymphocytosis in cerebro-spinal fluid.	Tabes (661).	871

CHART XIVc.—Disturbances of Vision

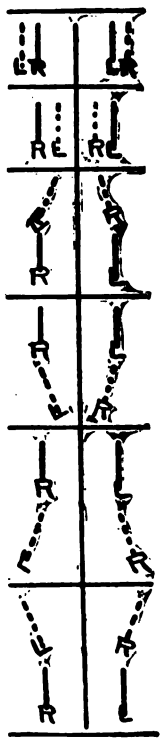
DIAGNOSTIC ANALYSIS OF SYMPTOMS

808 DISTURBANCES OF VISION (Continued)

818 Double vision. Diplopia (383-4). (Fig. 18) (Displacement of visual axis)

BINOCULAR — The images separate and come together again when the eyeballs are turned from one side to the other, or upward or downward and back again.

Secondary Deviation of Sound Eye (29)	Displacement of Visual Axis (28)	Limitation of Motion	Position of False Image (See 28)	Diagnosis	
Inward.	Inward. Strabismus convergens.	Outward.	On the same side as the affected eye.	External Rectus.	872
Outward.	Outward. Strabismus divergens.	Inward	On the opposite side to the affected eye.	Internal Rectus.	873
Upward.	Downward. Strabismus deorsum vergens, slightly divergens.	Upward and somewhat inward.	Above and on opposite side to the affected eye, image tilted top inward.	Superior Rectus.	874
Downward.	Upward. Strabismus sursum vergens, slightly divergens.	Downward and somewhat inward.	Below and on opposite side to the affected eye, image tilted top outward.	Inferior Rectus.	875
Down ward and inward.	None or slightly upward and inward. Strabismus sursum vergens, slightly convergens.	Rotation downward and somewhat outward.	Below and on same side as the affected eye, image tilted top inward.	Superior Oblique.	876
Upward and inward.	None or slightly downward and inward. Strabismus deorsum vergens, slightly convergens.	Rotation upward and somewhat outward.	Above and on same side as the affected eye, image tilted top outward.	Inferior Oblique.	877



The images do not separate and come together again as eyeballs are turned. — Absent. May be variable. The limitation of motion and the position of the false image are the reverse of those in paralysis. There may be present some irritation, especially in the nose or teeth, which would cause a reflex spasm. The spasm is usually more transient than a paralysis. The muscles usually affected are the internal rectus and the inferior oblique. — Spasm of the ocular muscles. 878

The whole eyeball can be seen to be displaced. — Displacement of eyeball. 879

MONOCULAR

- No changes visible in eye. — Hysterical symptoms (415) are present. — Hysterical diplopia. 880
- Changes visible in eye.
  - Two openings can be seen in pupil. — Double pupillary opening. 881
  - By oblique illumination the lens can be seen to be opaque in patches. — Cataract 882
  - Examination shows dislocation of lens, or detachment, or tumor, of the retina. — Dislocation of lens or retina. 883
  - Examination shows astigmatism and an irregular contour of the cornea. — Irregularities of cornea. 884

819 Conjugate deviation of eyeballs.

- Associated with other symptoms of lesions in the pons. Eyes turned away from the side of the lesion. Deviation is usually not present when the eyeballs are at rest. A vertical deviation of the eyeballs occurs very rarely. It is associated with a lesion of the corpora quadrigemina. (1294.) — Lesion near the anterior portion of the pons, cephalad to the abducens nucleus, involving posterior longitudinal bundle. 885
- Associated with other symptoms of lesions of the brain above the pons.
  - Eyes turned to the side of the lesion. — Paralytic lesion in almost any part of brain, especially, in posterior part of frontal lobe. 886
  - Eyes turned away from the side of the lesion. — Irritative lesion in cerebral cortex. 887

CHART XIVd

Abnormalities of Pupil and Optic Papilla

Comprising Numbers 820 to 821 and 890 to 893 on left side of Chart
and 894 to 915 on right margin

	DIAGNOSTIC SYMPTOMS AND TESTS					ABSTI
820 ABNORMALITY OF PUPIL.	Disordered pupillary reflex to light and accommodation (330-1). Mydriasis, miosis or unequal pupils (339-41).					These phenomena occur in t Their significance has bee
	890 The hemiopic pupillary reflex (?). (26).	Bitemporal hemianopia (362, 1335).				Choked disc. Symptoms pr
		Homonymous hemianopia (362, 1337).				Often hemiplegia or paralys history of syphilis. Very
	891 The Argyll-Robertson phenomenon (437).	History of syphilis. Lymphocytosis in cerebro-spinal fluid. Positive Wassermann.				Ataxia. Absence of knee-jer Mental impairment. Blurre Rarely occurs. No ataxia. I
821 ABNORMALITY OF PAPILLA. Discovered by Ophthalmoscopic Examination.	892 Optic neuritis. Choked disc.	Bilateral.	Retinitis.	No marked symptoms of cerebral disease.		Albumen and casts in urine. Sugar in urine and in blood, Lead in urine. Examination of the blood condition of severe anemi Urine and blood normal.
			No retinitis.	Marked cerebral symptoms, especially headache.		Well marked history of trau paralysis. Increased size of head and be increased in size, the Retraction of head. Cere General convulsion or Jack lepsy is common. May paralysis. Reflexes u creased. Cerebellar, but no motor a
		Unilateral.	Local inflammation can usually be discovered by examining the			
	893 Optic atrophy.	Bilateral.	Secondary.		It may be the terminal stage of a neuritis a Traces of the active inflammation (old hem	
			Primary. No signs of a former inflammation.		Old age. Usually atheromatous arteries and Loss of knee-jerk. Miosis. Lightning pains. Unequal pupils. Impairment of speech. Tre Characteristic tremor or other symptoms of t	
		Unilateral.	Local inflammation or lesion can usually be discovered on careful			

3 OF SYMPTOMS

PTIC NEURITIS AND ATROPHY

SYMPTOMS	DIAGNOSIS	
ditions to be of much diagnostic importance. n Chart Vb.		
rminating in blindness. Often associated with acromegaly.	Tumor compressing the optic chiasm (854, 864-5, 1335-6).	894
nerves. Optic neuritis or symptoms of meningitis. At times a rant hemianopia in partial lesions of the geniculate bodies.	Lesion of contralateral optic tract or geniculate bodies (862-3, 1337).	895
g pains. Girdle sensation and tabetic cuirass.	Tabes (611, 829, 912). (Figs. 24-7).	896
praxia. Restlessness. Childishness. Uncontrollable.	Paresis (913, 1106).	897
esent. May be no mental impairment. Normal speech. No apraxia.	Syphilis (1205).	898
Headaches, especially in morning. Usually edema of some part of body. Dyspnoea on exertion and loss of strength.	Bright's Disease.	899
Progressive emaciation and loss of strength. Great thirst and polyuria. Large appetite. Dry skin.	Diabetes Mellitus (1174).	900
Blue line on gums. History of lead colic. Wrist-drop. History of exposure to lead poison.	Lead Poisoning (494, 576, 584, 716, 790, 989, 1053).	901
Dyspnoea on exertion and progressive weakness. Pallor of skin and mucous membranes.	Anemia or Leukemia.	902
History of syphilis. Argyll-Robertson pupillary reflex. Lymphocytosis in cerebro-spinal fluid. Positive Wassermann.	Syphilis (1205).	903
hich the nerve has been injured. Usually complicated with facial	Injury.	904
nd sutures open in the young. In adults, in whom the skull cannot greater and the optic neuritis and headache are more intense.	Hydrocephalus (961).	905
mphocytosis. Fever.	Meningitis (500, 605).	906
May or may not be fever. At times a latent period. Primary suppuration of bones of skull or elsewhere. Optic neuritis present in about 53% of cases.	Cerebral Abscess or Sinus Thrombosis (508).	907
No fever. Usually steady progression. Optic neuritis present in about 80% of all cases; almost invariably present in tumors in the posterior fossa and in those associated with internal hydrocephalus. Tumors in pituitary gland, corpus callosum and in the central convolutions, especially extra-cerebral tumors, often show no optic neuritis.	Cerebral Tumor (507, 578).	908
ellar fits.	Cerebellar Tumor or Abscess (607-8-53-86, 784, 1016, 1295).	909
ow any of the causes of neuritis mentioned above. exudates, etc.) can usually be seen.	Terminal stage of Optic Neuritis (869).	910
l tension.	Senile Optic Atrophy.	911
sturbance.	Tabes (829). (Figs. 24-7).	912
impairment. Restlessness. Unreasonableness. Childishness.	Paresis (1106).	913
n usually be discovered on careful examination.	Disseminated Sclerosis (666).	914
	Disease of the eyeball or orbit (1338).	915

CHART XIVe

Abnormalities of Hearing, Taste, and Smell

DIAGNOSTIC ANALYSIS OF SYMPTOMS

Diagnostic Symptoms and Tests					Abstract of Symptoms	Diagnosis	
809 DISORDERS OF HEARING	822 DEAFNESS ANAKUSIA (355)	WORDS AND SOUNDS	Usually unilateral. May be bilateral. A permanent symptom.	Bone conduction impaired. No facial paralysis.	Severe paroxysmal vertigo and tinnitus aurium.	Ménière's or Labyrinth disease (649, 685, 1019).	918
					No vertigo. May be heredity. Locomotor ataxia or disseminated sclerosis may be present.	Atrophy of auditory nerve.	919
				Bone conduction not impaired. May be associated with facial paralysis.	May be history of syphilis, symptoms of meningitis, symptoms of tumor at base, optic neuritis, etc.	Tumor or inflammation involving auditory nerve trunk.	920
					Disease of, or injury to, middle or outer ear; cerumen.	Lesion of ear.	921
			Usually bilateral. Very rarely unilateral, and then only a transitory symptom.		Associated with symptoms of lesion of the pons or crura cerebri.	Bilateral lesion of the lemniscus. (Fig. 20).	922
					Associated with symptoms of lesion of the cerebral cortex. Complete deafness does not always occur in a bilateral lesion of the temporal cortex.	Lesion of the temporal cortex on both sides. (Fig. 15).	923
					Hysterical symptoms (415). No symptoms of organic disease.	Hysterical deafness (1076).	924
			Deafness may be caused by lesions in the ear or auditory nerve or by bilateral lesions in the brain stem. There is reason to believe that the cortical center (or area) for hearing is in the anterior transverse tecporal convolution which is situated in the percular (upper) surfaec of the superior temporal. Innervation may bilateral.			Lesion of transverse temporal convolution.	924a
		WORDS ONLY. Sensory aphasia (223) is present.				Lesion of left superior temporal convolution. (Fig. 15).	925
	823 HYPERAKUSIA, OXYAKOIA OR PARAKUSIA (372, 389).				Hysterical symptoms are present.	Hysteria (1076).	926
					Inflammatory lesions of ear or its neighborhood are present.	Hyperemia of inner ear.	927
					Facial paralysis is present. Low notes are especially painful. Tinnitus aurium is present.	Facial paralysis (1333).	928

810 DISORDERS OF SMELL AND TASTE. Very little, if any, diagnostic significance can be attached to disturbances of smell and taste. The cortical area for smell seems to be in the cornu Ammonis; while that for taste is probably slightly posterior and external to the olfactory area; but no case has been recorded where loss of taste or smell has been proved to be due to any cortical lesion.

CHART XV

Perversion of Sensation: Pain and Vertigo ||

DIAGNOSTIC ANALYSIS OF SYMPTOMS

DISORDERS OF SENSATION—PERVERSION

Symptoms Analysed		Location of Pain	
930 PERVERSION OF SENSATION IN NERVOUS DISEASES (347).	931 PAIN (374).	933 PAIN IN NERVE Pain limited to the trunk and branches of one nerve in any part of the body, except that at the height of the attack, there may be a mild radiation of the pain into corresponding nerve of opposite side or into adjacent nerves. 934 PAIN IN HEAD. HEADACHES IN NERVOUS DISEASE After a careful examination with suitable instruments has proved the absence of glaucoma, iritis, muscular insufficiencies and other diseases of the eye, of the nose and its sinuses, of the teeth, of the ear, of the scalp (rheumatism) or of the cranial bones (periostitis, caries).	See Chart XV a.
		935 PAIN IN TRUNK IN NERVOUS DISEASE After a careful examination has proved the absence of Pott's disease, rheumatism of spine or trunk muscles, arthritis, disease of breast, pericarditis, pleurisy, aneurism, pleurodynia, periostitis, cancer and other tumors, colic (intestinal, uterine, biliary, renal) dyspepsia, pancreatitis, appendicitis, peritonitis, gastric ulcer, gastritis, enteritis, hernia, floating kidney, tubal pregnancy, pelvic inflammation, intestinal obstruction, etc.	See Chart XV b.
		936 PAIN IN EXTREMITIES IN NERVOUS DISEASE After a careful examination has proved the absence of any disease of the bones, muscles, joints, blood vessels or skin of the arms and legs. Flat foot must be excluded.	See Chart XV c.
	932 VERTIGO (392).		See Chart XV d.

CHART XVa

Pain in Nerve, Pain in the Head, Headache

Comprising Numbers 933 to 934 on the left side of margin
and 937 to 968 on the right margin

DIAGNOSTIC SYMPTOMS AND TESTS

933 PAIN IN NERVE			The differential diagnosis between neuritis and neuralgia cannot always be made clinically. The diagnosis is aided by the experience that certain nerves, such as the sciatic, are more prone to neuritis; while others, such as the trigeminal, are more prone to neuralgia. (Figs. 33, 38).	Paroxysmal pain with free intervals.	Never any motor paralysis or persistent anesthesia or loss of reflexes.
				Continuous pain with exacerbations.	May be motor paralysis or anesthesia or loss of reflexes or all combined.
934 PAIN IN THE HEAD	APYREXIA	LOCALIZED PAIN	A history of neurotic heredity or other evidence of a neuropathic predisposition, congenital or acquired, is common. Pain is unilateral and is increased by movement and by exposure to cold or wind and is sometimes associated with muscle spasm. Vasomotor and trophic disturbances are often present.	Pain limited to the whole or a portion of the trunk and distribution of the trigeminal (prosopalgia) or occipital nerves. Diseases of the eye, the nose and its sinuses, the teeth, the ear, the scalp and the bones must first be excluded. (For the diagnosis between neuritis and neuralgia see 933).	The pain is felt above the eye in the fo tender. If tension of eyeball be increa The pain is felt below the eye in the ch The pain is felt in the lower jaw and it The pain is felt in two or three of the sit The pain is momentary in duration and The pain is felt in the occipital region i common and early symptom in neura
				Pain strictly limited to one lateral half of the head.	Periodical attacks (often occurring at m crania angio-paralytica) or pallor, cok Motor or sensory aphasia may be pre age and often shows a direct inherita
				Pain as if nail were being driven through the skull.	Pain of great intensity in
			Pain, nocturnal, in small area and spreading.	History or other evidence of syphilis (not always obtainable).	Pain may be felt at any time but is wor quent. Cranium is often tender at poi
			Pain localized in small area.	Disease exists in organs within the head or body.	Frontal headache may be due to gastric d to pelvic disease. These referred pai however, should not be attached to th
		DIFFUSE PAIN	Evidence of poisoning.	Exogenetic.	Occurs after the ingestion of narcotics. Occurs as the result of breathing for hou
				Auto-genetic.	Occurs as the result of constipation, es Occurs in Bright's disease, usually is wo
			Evidence of circulatory disorder.	Cerebral hyperemia.	Headache with fulness and throbbing in may be followed by a cerebral hemorrh
				Cerebral anemia.	Headache, most commonly at vertex, wi posture. In this, as in other forms o
			Evidence of nervous exhaustion.	Headache associated with phobias and tremors and insomnia and pressure within the skull, especially pressure in occipital and
			Evidence of serious brain disease. Headache often associated with vomiting, or vertigo, or both.	Optic neuritis or choked disc.	Progressive symptoms, motor or sensory over the seat of the lesion. Lumbar Cerebellar, without motor, ataxia is pre
				May follow traumatism.	Intractable, incurable, more or less cons or stretching of the dura mater by tu
			Chronic headache. Pain constant with exacerbations.	Evidence of rheumatism elsewhere.	Diffuse pain and tenderness of scalp. Ps
	PYREXIA.		Evidence of infection.	Headache.	Temporary. Occurs during th Permanent. Occurs throughou Suppuration else
	HYPER-PYREXIA.		Evidence of exposure to high temperature.		History of exposure to high temperatur

CHART XVb

Pain in Trunk

Comprising Numbers 935 on left side of Chart
and 970 to 991 on right margin

CHART XVc

Pain in Extremities

Comprising Numbers 936 on left side of Chart
and 995 to 1012 on right margin

DIAGNOSTIC SYMPTOMS AND TESTS			ABSTRACT OF
936 PAIN IN EXTREMITIES IN NERVOUS DISEASES.	Unilateral. Many of these forms of neuritis may be associated with, or precede, or follow a rash of herpes: herpetic ganglionitis. (Figs. 33, 38).	Pain in arm.	Pain radiates along one or all of the nerves of the arm. Tender po other points where nerves are superficial. Vaso-motor disturba paralysis; but movements of arm are impaired by the pain. Tum pressure on nerves, must be carefully excluded.
		Pain limited to the trunk and distribution of the sciatic, anterior crural or obturator nerve.	Pain shooting along the trunk, or over small areas in the distribu ysis, but the pain may prevent motion. Patient holds knee of t body to the opposite side and bears his weight on the healthy leg. trochanter major (trochanteric point) and in popliteal space (p and, then, there may be decided muscular weakness and atrophy. rectal examination for any possible pressure upon the nerve sho in sciatic neuritis.
			Pain along the trunk and distribution of the anterior crural nerve ankle. Tender points on anterior aspect of the hip joint, inner paralysed and atrophied and knee-jerk lost and anesthesia may b be secondary to diabetes and injury. There may be an eruption
			Pain along inner side of thigh, along course of obturator nerve, af neuralgia and is usually associated with paralysis of the adduc
		Pain limited to outer surface of thigh.	Pain is associated with paresthesiae (especially numbness and ting flat foot or weakened arch. The paresthesiae are more charac absent.
		Pain in a joint.	Pain in a joint, usually the knee-joint, increased on motion. The dence of any disease of the joint. Many hysterical symptoms
		Pain at insertion of Achilles' tendon.	Severe pain at insertion of Achilles' tendon on walking and stand
		Pain in heel.	Pain in lower surface of heel, especially when walking or standing. gical removal of the sub-calcaneal bursa, or of exostoses, others
		Pain in toe.	Pain in the metatarso-phalangeal joint, especially of the fourth toe, is lowered from "breaking" of the arch transversely.
		Burning pains.	Intense burning pain, usually in foot or hand, often associated with nerve injury which is not complete. At times due to flat foot.
	Bilateral.	With girdle pains, and lumbar puncture gives lymphocytosis.	With Romberg's symptom, Argyll-Robertson's phenomenon, ataxia, lymphocytosis and lightning pains over small areas in legs, supe
			With pain and rigidity in back and in extremities. Exaggerated re cocytosis or lymphocytosis in cerebro-spinal fluid. In cases in dolorosa" may result.
		With anesthesia.	Steadily progressive motor and sensory symptoms, at first mainly fluid. Brown-Sequard's paralysis (432).
			Motor paralysis and anesthesia over whole of both legs, except in peripheral and organic reflexes. Muscular atrophy and trophic in lower back and radiating into legs.
			Motor and sensory paralysis commencing at the distal end of extren and tenderness. The disease usually commences with pain or
		With dissociation of sensation.	Pain and paresthesiae, analgesia and thermic anesthesia without symptoms are usually limited to arms with symptoms of spastic but are milder.
		With vaso-motor disturbances.	Extreme pain in soles of feet associated with redness and swelling a Flat foot must be excluded.
			Pallor and coldness of fingers and toes followed by cyanosis and con In extreme cases a larger or smaller slough forms and is cast off.
		With fat.	Marked increase in fat, either diffuse or in separate tumors, in arm ated with it, and the fatty masses are tender, especially in the

F SYMPTOMS

ITIES

PTOMS	DIAGNOSIS	
in supra-clavicular fossa, in axilla, at head of radius and at Fibrillary contractions at times occur. There is no motor t base of neck and in axilla, and a cervical rib (557), causing	Cervico-brachial Neuralgia or Neuritis of Ulnar, Median, Radial, etc., according to the distribution of the pain.	995
of the sciatic nerve. Little, if any, anesthesia or motor paral- ffected side semi-flexed, thigh slightly abducted, inclines his nder points over the sciatic notch (gluteal point), above the eal point). In neuritis, the nerve, wherever felt, is tender, atica is much more frequently a neuritis than a neuralgia. A lways be made. The tendo Achillis reflex is often abolished	Sciatica (720).	996
be anterior surface of the thigh and inner surface of leg to the of knee and at internal malleolus. Extensors of thigh may be anterior surface of thigh and inner side of leg in neuritis. May erpes along the course of the nerve.	Crural Neuralgia or Neuritis.	997
ernia and other diseases have been excluded. A rare form of	Obturator Neuralgia.	998
and is probably always associated with, and is caused by, ic of this disease than is the pain. which is often entirely	Meralgia Paresthetica.	999
is much more sensitive than the articular surface. No evi-).	Arthralgia or Hysterical Joint.	1000
May follow gonorrhea, malaria, gout, broken arches or injury.	Achillodynia.	1001
e cases are cured by anti-rheumatic medicine, others by sur- upporting the weakened arches.	Talalgia or Calcanodynia.	1002
lly following an injury. Usually occurs in women. The joint	Metatarsalgia or Morton's Toe.	1003
hidrosis and vaso-motor disturbances. Usually caused by	Causalgia or Thermalgia.	1003a
ry of syphilis usually, always loss of knee-jerk, cerebro-spinal l and deep, often followed by hyperalgesia over same area.	Tabes. Neuralgic stage (661).	1004
s. No ataxia. No Argyll-Robertson's phenomenon. Leu- the cord is secondarily compressed or involved "paraplegia	Spinal Meningitis (605, 974, 1203-4).	1005
teral, later bilateral. Increased pressure of cerebro-spinal	Spinal Tumor (509, 828, 839-44, 975).	1006
cases the domain of the anterior crural nerves. Abolition of rbances. Anesthesia in perineum and genitals and much pain	Lesions of Cauda Equina (487). (Fig. 29.)	1007
and extending towards body. Muscular weakness, atrophy hesiae in toes and fingers and often with fever.	Multiple Neuritis (488).	1008
le anesthesia. Trophic disturbances and mutilations. These plegia in legs. The pains often resemble the pains of tabes,	Syringomyelia (553, 693, 840-2, 1152, 1170, 1187, 1370-2).	1009
ater with pallor, shrinking and wrinkling of the same parts.	Erythromelalgia (1198).	1010
on; so that fingers and toes become purplish and even black.	Raynaud's Disease (1195).	1011
d legs, but not elsewhere. There is considerable pain associ- stages when they are forming.	Adiposis Dolorosa. Dercum's Disease (1175).	1012

CHART XV d

Vertigo

Comprising Numbers 932 on left side of Chart
and 1015 to 1034 on right margin

DIAGNOSTIC SYMPTOMS AND TESTS			ABSTRACT OF S
	Motor Ataxia is present.		In these cases the vertigo is not a prominent symptom. In some cases, in of falling and fears that he will fall and experiences some vertigo; while lesions in the brain stem and elsewhere. The diagnosis is made from th
	Cerebellar Ataxia is present.		Any disease of the cerebellum, especially tumors, may cause vertigo, whic of the hemispheres. The diagnosis is made from the absence of paralysi and, in tumors, the optic neuritis and failure of sight.
	Crossed Paralysis.		Lesions of the brain stem may involve the tracts from the cerebellum and made by the motor or sensory paralysis or both, which occurs in the for paralysis in the domain of the cranial nerves (crossed paralysis, etc.).
	Vertigo on movement of head.		Cysts and tumors suspended free in the fourth ventricle cause intense dizz drome). Except for this symptom the diagnosis is extremely difficult or i the position in which the head is held. Choked disc is common.
	Deafness and symptoms of aural disease.		A steadily, progressive deafness of one ear associated with tinnitus in that may throw patient to the ground. Raising the head from the ground ma or loss of bone conduction and loss of power of hearing high notes are usu the paroxysmal attacks. Suppurative and other diseases of the ear may when the ear is completely deaf, but then may commence in the other ea may cause vertigo by affecting the semi-circular canals or vestibular ne laesa). It is difficult to draw the line between these cases of aural ve cover all these conditions. Strictly speaking Ménière's disease applies onl flammation of the labyrinth causing vertigo is called Voltoni's disease.
	Diplopia and symptoms of ocular disease.		Double vision and weakness of ocular muscles and eye strain may cause ve is relieved by closing the defective eye, even when it is not caused by t
932 VERTIGO (392)	Symptoms of circulatory or digestive disturbances.	Position and moving.	When patient's head is bent down for a long time and then is suddenly r ences vertigo. A blow on the head will cause vertigo, probably in conse of back of head or moving head quickly may cause vertigo. A similar rent to the head.
		Exhaustion.	Great weakness, especially in the convalescence from disease, is a common
		Digestive disorders.	When, in consequence of the congestion due to digestive disorders, the por are anemic. These digestive disorders may also produce abnormal che diagnosis is made by the presence of the digestive disorder and by the
		Cardiac and hemic disease.	In all forms of cardiac disease the brain may receive an insufficient and irre frequent in aortic disease. The diagnosis is made from the presence of to the altered quality than quantity of the blood supply (1030).
		Atheromatous arteries.	Atheromatous arteries interfere with the normal blood supply both as to a cause vertigo. This is especially common in elderly people. The diagnosi usually, an increased arterial tension.
		High blood tension.	Fulness of head, headache, mental confusion, tinnitus aurium, palpitation
		Apoplexy.	Vertigo is a common initial symptom of apoplexy of all forms (cerebral he rhage) and may be the only symptom of a slight attack. Usually the sequ
		Epilepsy.	Vertigo may constitute the aura which may or may not be followed by a tacks. In some cases a severe subjective sensation of vertigo, frequently attack. Vertigo is not an uncommon symptom in the interval between th
		Migraine.	Vertigo may be the initial symptom or may accompany an attack of migrain makes the diagnosis plain.
	Toxic.		Abnormal conditions of the blood, as in the early stages of infectious dise Various toxic substances, such as tobacco, alcohol, coffee, morphia, quinine, tion of the cerebral or cerebellar cortex. The diagnosis is made by the p vertigo. A disease endemic in Switzerland and occurring only in men working in h ness of vision, ptosis, often diplopia without strabismus, and a paralysis Pain in back of neck. Attack lasts a few minutes.
	Symptoms of cerebral disease (headache, etc.).	Organic.	In addition to apoplexy, any irritation of the meninges (tumors, local lesio associated with severe vertigo, especially on change of position. Tumor mitted pressure on the cerebellum, or, when situated in the frontal lobe, diagnosis is made by the numerous other symptoms of these diseases: c associated with the vertigo, which is less severe in the recumbent posture.
		Functional.	Vertigo is not an uncommon symptom in those functional nervous diseases chronic, such as neurasthenia, the traumatic neuroses and hysteria. T This vertigo is never very severe and often rather resemble syncopal att

TOMS	DIAGNOSIS	
equence of the incoördination, the patient is in danger ther cases the vertigo may be the direct result of the sence of motor ataxia.	Tabes, Disseminated Sclerosis and other diseases with ataxia.	1015
more permanent in lesions of the vermis than in those presence of cerebellar ataxia, headache, and vomiting	Cerebellar Disease (392, 607-8-47-86, 784, 1295).	1016
ataxia and, less frequently, vertigo. The diagnosis is hemiplegia with increased refleres, and also by local 19-22).	Lesions of the brain stem (460, 535-46, 656, 832, 1290-7, 1321-4, 1387-90-4-6, 1400-8).	1017
ely, or mainly, when head is moved. (Brun's syn- sible. The vertigo may vary greatly in intensity with	Lesions within the fourth ventricle (Fig. 19).	1018
and with paroxysmal attacks of severe vertigo which vomiting. Attacks vary in severity. Impairment present. Vertigo is usually entirely absent between present, but usually are not. Disease usually ceases most any disease or functional disturbance of the ear directly or indirectly (aural vertigo or vertigo ab aure and Ménière's disease, which latter is often used to cases of hemorrhage into the semi-circular canals. In-	Ménière's Disease. Voltoni's Disease. Aural Vertigo. Vertigo ab aure laesa (649,685, 918).	1019
Occurs sometimes on railway trains. The vertigo ia alone.	Ocular Vertigo. Vertigo ab oculo laeso (648).	1020
when patient's body is rotated rapidly, he experi- of vaso-motor reflex disturbance. Lying on one side may result from the application of a galvanic cur-	Acute Cerebral Anemia.	1021
both of vertigo and ataxia.	Exhaustion Vertigo.	1022
lation is engorged with blood, the cerebral vessels stances which may produce a toxic vertigo. The the vertigo when the indigestion is cured.	Acute Cerebral Anemia from digestive disorders, internal hemorrhage, etc.	1023
ply of blood and vertigo may result. This is most disease. In hemic diseases the vertigo is due rather	Chronic Cerebral Anemia from blood and cardiac diseases.	1024
and as to uniformity of distribution and hence may me from the presence of atheromatous arteries with,	Chronic Cerebral Anemia from atheromatous arteries (syphilis).	1025
, dyspnoea on exertion, and high blood tension.	Cerebral Congestion.	1026
, embolism and thrombosis, and meningeal hemor- other symptoms makes the diagnosis clear.	Apoplexy (504).	1027
k. The diagnosis is made from the epileptic at- by vomiting, may be the equivalent of an epileptic , and may continue during minutes or hours.	Epilepsy (575, 1061, 1073).	1028
hemicrania, the much more prominent symptom,	Migraine (849-58, 950).	1029
in leukemia, melanemia, gout, diabetes, etc.	Toxic Vertigo (1024).	1030
cause vertigo, probably by affecting the circula- ingestion of the substances before each attack of	Drug Vertigo.	1031
ables. It consists in attacks of vertigo, with dim- function or act of the arms, simulating hysteria.	Gerlier's Vertigo. Vertige Paralysant.	1032
especially inflammations and syphilitic lesions) is both by irritation of the meninges and by trans- irritation of the cerebro-cerebellar tract. The , vomiting, slow pulse, etc., which are frequently	Cerebral Meningitis and Tumor (Syphilis) (508, 536-42).	1033
are the result of psychic traumata, acute and ential diagnosis of these is made in other charts.	Neurasthenia. Traumatic Neuroses and Hysteria (1074-7).	1034

CHART XVI

Disorders of Cerebral Activity

DIAGNOSTIC ANALYSIS OF SYMPTOMS

Symptoms Analysed	Alterations in Mentality	
1036 Disordered Mentality.	1037 Coma.	See Chart XVIa.
	1038 Pseudo-Coma.	See Chart XVIb.
	1039 Double Personality.	
	1040 Weakened Mentality.	
	1041 Insanity.	See Chart XVIc.

CHART XVIa

Coma

Comprising Numbers 1037 on left side of Chart
and 1042 to 1070 on right margin

COMA OR SEMI-COMA (205,745).

See also pseudo-coma, (1088).

History, or other evidence of recent injury to head.	**Convulsions rare.**	Pupils usually contracted and respond feebly to light. Patient may be co Symptoms follow
		Pupils dilated, often unequal, and usually do not respond to light. Symptoms usually usual symptoms.
	Convulsions are frequently present.	The symptoms are those of a local meningitis (507, 1049) o the coma are common. There may be local symptoms, bo may be edema of eyelids and conjunctivae, choked discs, p
		Retraction of neck and opisthotonos. Fever, headache, del irritation followed by those of paralysis. Pulse usually
	Sporadic.	An acute disease characterized by *general* symptoms; such cephalitis has been subdivided into a number of special
	Epidemic.	The disease often follows an attack of influenza, and is of more of the motor cranial nuclei, especially of the motor psychotic form (delirium). No pain. Moderate fever. symptoms of paralysis agitans. The mortality from the
No preceding injury or disease.		After uncertain prodromata, coma and paralysis with fever polioencephalitis superior hemorrhagica acuta) or may be
		Headache, vertigo and vomiting. Often mild delirium. A re general and resemble those of a rapidly growing tumor. C
		Headache, vertigo and vomiting. May be a history of form tumors at the base are more likely to cause paralysis of
History of a previous illness, of which the coma is only one symptom, and often the terminal one; or the presence of an inflammation of the scalp (erysipelas, suppuration), or of the bones of the skull (caries and especially suppuration of the bones of the ear).		Hemiplegia or diplegia occurring in early infancy is comm fect. Epileptiform convulsions, unilateral or bilateral,
		Progressive mental impairment, childishness, restlessness, words, syllables and letters are left out and letters doub
		History of lead poisoning, of lead colic, of wrist-drop, etc.
		Intention tremor. Scanning speech. Many motor and senso
		Headache, increasing fever, polyglandular enlargement, dif fly by which trypanosomes enter the blood and multiply t
History or other evidence of poisoning.	**Convulsions rare.**	Patient can usually be aroused from his coma sufficently to of alcoholic abuse. Alcohol may, possibly, be found in
	Convulsions absent.	Pupils are contracted and do not respond to light. Patie taken morphine or opium.
		Patient is in a confined space or room in which there is a
Evidence of a cardiac inadequacy.	**Often a slight spasm or rigidity during the attack.**	Sudden attack of coma with pallor and weak or absent ca small, extremities cold, restlessness, yawning. Low arte
		Sudden intermission of heart beat during a considerable fr
Sudden attack of coma of short duration with or without a convulsion. Usually a history of similar attacks and often of remote injury.	**Convulsions almost always present and are usually the most striking symptom of the disease, but not so characteristic as unconsciousness, which is at times the only symptom of the attack.**	The attack usually commences with a convulsion, as descril tion, sometimes trivial, sometimes a deed of violence (po unconsciousness and no convulsion and either no action c replaces the convulsive attack and is called the "psychic wander about and take journeys and are lost to their fri essential characteristic of an epileptic attack is the comp quently he is irritable and shows some mental weakness peutically. On the other hand, many epileptics have atta
		The attack is altogether similar to the major attack of epile distended abdomen, foul smelling feces, vomiting, diarrho
		Sudden attack of tickling and burning in larynx, suffocati
Sudden attack of unilateral paralysis. Rarely the paralysis comes on slowly, steadily increasing during hours or days: "Ingravescent apoplexy." In such cases the coma may be slight or absent.	**Convulsions are rare, but both tonic and clonic spasms may occur, involving one-half of the body when the lesion is cortical, or involving both sides of the body when the hemorrhage occurs in the brain stem (460) or ruptures into a ventricle.**	The coma comes on instantly or in the course of a few hou flaccid paralysis in the form of hemiplegia. The bilaterall laryngeal, respiratory, abdominal, micturition, defecation ent from the start. The other reflexes may, during the c may die in coma or the coma may pass away after severa paralysed. Tongue protruded towards the paralysed side, disappears, but which may be permanent, in which latte Improvement usually begins in the face, next in leg and tractures appear in arm and extensor contractures in le hand. Some mental impairment persists. Local symptor lesion may cause an exacerbation of the symptoms in the
Albumen and casts, or sugar, or all three, in urine.	**No convulsions.**	Patient emaciated. Acetone odor of breath. Pulse is smal
	Convulsions usually.	Onset usually gradual. Some edema, cyanosis, restlessness, tains albumen and casts.
Decided fever. / **Hyperpyrexia.**	**Convulsions frequently.**	Occurs at the onset of acute infections, especially in childr of this nature, but in these cases convulsions are rare, an
		History or evidence of exposure to great heat. Absence of symptoms (paralytic) occasionally occur.

residence in Also malarial malarial regions. during and after

CHART XVIb

Pseudo-coma, Double Personality and Weakened Mentality

Comprising Numbers 1038 to 1040 on left side of Chart
and 1071 to 1077 on right margin

CHART XVIc

Insanity

Comprising Numbers 1041 and 1078 to 1082 on left side of Chart
and 1083 to 1120 on right margin

1041 INSANITY (110, 130, 155).

- **DEFECT IN INTELLIGENCE**
  - **1078 Amentia (211).** More or less complete. (A mental defect either congenital or manifested in infancy).
    - A condition in which the mind has not developed with advancing age, due to a disease of the brain, either congenital or acquired in infancy. Besides the mental defect, these patients often present many and various physical defects and deformities such as: deformed skull, posterior hydrocephalus, high palatine arch, coarse body, deformed ears, etc. The amentia may be either general or partial, and some of its slight degrees may be due in part to defective training.
      - Patients show little or no intelligence. Are unab walk. Usually soil themselves with urine and requiring intelligence and are incapable of sustai outbreaks of anger. Many of them are undersiz
      - Patients can talk and are more or less cleanly in gence. They are incapable of much education. is common and sexual instinct is often strongly
      - Patients show a degree of intelligence approaching cation, even though in general amiable. Some cl Tartar-like expression of the countenance, slant may be due in some cases to poison, such as wi
      - Certain feeble-minded persons seem incapable of a
  - **1079 Dementia (212)** More or less complete. (A loss of mentality, previously acquired, first manifested after infancy).
    - A condition in which the mind has developed to a certain, even a high, degree of intelligence and then, in consequence of disease of the brain (functional or organic), all mental development has not only ceased, but there has been a distinct retrogression which may go on to a complete loss of intelligence. Memory, emotions and interest are all lost. Patient becomes apathetic, reacts to no stimulation, soils himself and does not even eat.
      - Occurring in youth, at puberty or before 25 or 30 years.
        - Complete apathy, coming on more or less acutely wi and will respond to no stimulation. Appears to b reflexes are exaggerated. The course of the dise
        - Partial apathy. Patients are dull and stupid but c is an absence of emotions and of interest in anyt They perform frequently spontaneous, impulsive, of a phrase which they have just heard or spoke The varieties under this head merge into each ot
      - Usually occurring in adult life after 25 years, but may occur in youth.
        - History of alcoholism and usually associated with and space.
        - History of alcoholism extending over many years. amounts of alcohol. The symptoms at times resen
        - History of very numerous epileptic seizures. Gradu
        - History of a previous psychosis which has graduall (apathetic dementia) but some cases show great
        - History of syphilis. Lumbar puncture shows globu bility for continuous mental concentration, reckless cheerfulness, in spite of the illness which patient r No paralysis, but much paresis and especially apr more rarely with various forms of spinal sclerosis
      - Occurring in old age.
        - Associated with physical weakness and with athero loss of memory, especially for recent events, wit
- **1080 Hallucinations are abundant and dominant. Hallucinatory Insanity (213-7), or Delirium.**
  - A condition in which the patient is constantly receiving false perceptions from his different senses: either visual, auditory, olfactory, gustatory, tactile or painful, or from several or all combined. Associated with this is always a certain degree of impairment of consciousness, which weakens his judgment and does not permit him to decide that these hallucinations are false.
    - Patient is overwhelmed by a large number of that of those about him, and especially dou
    - History of alcoholism. Patient's hallucinatio dent. Great fright. Violent attempt to es
    - Many other poisons besides alcohol; either ex a mild hallucinatory insanity or delirium, w
    - Disease commences with fever, headache, daz
- **1081 Delusions are present and dominant. Delusional Insanity (215)**
  - A condition in which the patient has formed a false judgment about things which concern him. The basis of these false judgments is partly a congenitally defective brain and partly hallucinations. Associated with these delusions there is always present a varying degree of impairment of intelligence, which prevents the patient from recognizing the falseness of the delusion when evidence is presented to him which would be adequate for a normal man; although many of these patients in their own way reason shrewdly. These delusions lead to irrational conduct on the part of the patient which would not be irrational were the delusions true.
    - History of alcoholism. Patient has formed, wives are unfaithful. Patients often act vi
    - Patient has delusions upon which he bases his upon them. Curable in most cases.
    - Patient has a number of delusions, unsystematized or nearly so but which are strong enough to influence his conduct and bearing. Curable in most cases.
    - Patient has many delusions which are often are woven in with the delusions in the att thus systematized and some wild theory is markable person, because so many persons happening is to injure him or to try his ch
- **1082 An exaggerated emotional state is the dominant symptom. Emotional Insanity (204).** The insanities of the neuroses have been considered under epilepsy, hysteria and hypochondriasis.
  - Exaggeration of the sometimes natural feeling of sadness or discouragement with life. An hereditary predisposition is frequent.
    - Patient is constantly in a depressed and painful fra by the patient to explain the melancholy; the very prone to suicide. Their circulation and thei
  - Exaggeration of the natural feeling of joyousness.
    - Patients are constantly in a joyous and excited mo tomatic and due to delusions (1114). Mania is
  - Alternation of mania and melancholia.
    - Alterations at long intervals of mania and melanc
    - Attacks of excitement or of depression may recur Kraepelin has incorporated all of these forms un

CHART XVII

Trophic and Sympathetic Disorders

DIAGNOSTIC ANALYSIS OF SYMPTOMS

TROPHIC DISORDERS AND DISORDERS OF THE SYMPATHETIC SYSTEM

Tissues Involved	Symptoms Aanalysed	
1121 Trophic Lesions.	1123 Muscular Tissue.	See Chart XVIIa.
	1124 Cutaneous and Sub-Cutaneous Tissue.	See Chart XVIIb.
	1125 Fatty Tissue. 1126 Bone Tissue. 1127 Joint Disease. 1128 Other Trophic Lesions.	See Chart XVIIc.
1122 Disorders of the Sympathetic System.	1129 Ganglionic Disorders. 1130 Vaso-Motor Disorders.	See Chart XVIId.

CHART XVIIa

Muscular Atrophy and Hypertrophy

Comprising Numbers 1123, 1131 and 1132 on left side of Chart
and 1146 to 1158 on right margin

DIAGNOSTIC ANALYSI

MUSCULAR ATROPHY A

DIAGNOSTIC SYMPTOMS AND TESTS

ABSTRACT OF S

1123 MUSCULAR TISSUE.	1131 ATROPHY.	Atrophy is relatively rapid in onset and usually great in degree.	Muscular atrophy. Lesion in peripheral motor neurons.	Acute and subacute course (inflammatory lesions).	Paralysis is the primary symptom and atrophy is secondary to it.	
				Chronic course (degenerative lesions).	Atrophy is the primary symptom and the paralysis is secondary to, and consequent upon, it.	
				Associated with chronic joint disease, especi be found.		
			Muscular atrophy and hypertrophy combined. Lesion in muscles.	Muscles of face (Landouzy-Dejerine type), o are first affected. Some muscles apparentl hypertrophied, with increase of interstitial		
		Atrophy is very slow of onset and often slight in degree.	Lesion in central motor neurons.	Very slow course.	Paralysis is primary and atrophy is secondary.	
	1132 HYPERTROPHY	Increased or normal strength.	No lesion.	Muscular fibers normal. A true hypertrophy.	The hypertrophy is the re The hypertrophy is due to heredity.	
		Decreased strength.	Lesion in muscles.	Calf muscles, infra-spinatus, deltoid and some muscles are both weak and atrophied. No some hypertrophied and much interstitial f affected.		

MPTOMS

RTROPHY

S		DIAGNOSIS	
	History of injury, wound or scar.	Injury of nerve (489, 824).	1146
	Limited to distribution of one nerve (simple neuritis) or many nerves (multiple neuritis). Usually associated with sensory symptoms: pain and anesthesia, nerve and muscle tenderness.	Neuritis (488-92, 824, 933, 940-9, 1173). (Figs. 33, 38.)	1147
or par- rical of tion.	Groups of muscles attacked not corresponding to the distribution of any nerve. No sensory symptoms, except some pain at onset in back, joints and muscles. Very rarely nerve and muscle tenderness. Globulin and lymphocytosis in cerebro-spinal fluid in acute stage.	Acute anterior poliomyelitis (495, 791). (Figs. 26-7.)	1148
	Atrophy affects either the arms or the legs. Sensory and other symptoms of myelitis are present. Organic reflexes are more or less disordered. Superficial and deep reflexes are abolished in the paralysed area.	Myelitis of Cervical or Lumbar Enlargement (485, 550).	1149
	Atrophy commences in the small muscles of hands, or muscles of shoulder girdle, and extends and is associated with fibrillary contractions. Mild spastic paraplegia (525, 800) in legs.	Amyotrophic lateral sclerosis (547-8, 695, 800). (Figs. 26-7.)	1150
on of the excitabil- o reaction ration.	Atrophy affects the muscles of tongue and lips and is associated with fibrillary contractions. Mild spastic paraplegia (525, 800) in legs.	Chronic bulbar paralysis (546, 694). (Figs. 21-2.)	1151
	Atrophy affects the hands usually. Is associated with dissociation of sensation and often with ulceration and mutilation.	Syringomyelia (553, 693, 840-2, 1009, 1170, 1370-2).	1152
nkylosis.	Many of these cases are neuritic, but in some no neuritis can	Arthritic atrophy.	1153
er girdle (Erb's juvenile type), or of legs (pseudo-hypertrophic form) hied. Excised muscle fibers show degeneration; some atrophied, a few fibrillary contractions.		Muscular dystrophies (477, 787, 1158).	1154
phy is due to disuse. rical reac- degenera-	The reflexes are exaggerated. Ankle-clonus and Babinski are present when legs are affected, unless prevented by contractures.	A paralysis of long standing, especially one from infancy.	1155
h exercise, and is indicative of increased power.		Strong man or athlete.	1156
sm, occurring at the commencement of voluntary motion. Strong		Thomsen's disease (611).	1157
les appear large, but are weak; a false or apparent hypertrophy. Other ontraction. Excised muscle fibers show degeneration: some atrophied, ourse. All muscles are finally atrophied. Legs are early and mainly		Pseudo-hypertrophic paralysis (500) and the muscular dystrophies (1154).	1158

CHART XVIIb

Cutaneous and Sub-cutaneous Trophic Disorders

Comprising Numbers 1124 and 1133 to 1136 on left side of Chart
and 1160 to 1173 on right margin

ENDOCRINOPATHIES

A number of diseases in the following charts have been proved to be due to abnormalities of the internal secretions of certain ductless glands.

Thyroid Gland	EXCESSIVE SECRETION or HYPERTHYROIDISM	Exophthalmic Goiter.	See 1193
	DIMINISHED SECRETION or HYPOTHYROIDISM	In adult. Myxoedema	See 1163
		In Infant. Cretinism	See 1164
Parathyroid Gland	DIMINISHED SECRETION	Some forms of Tetany	See 614
Pituitary Body	EXCESSIVE SECRETION or HYPERPITUITARISM	Acromegaly	See 1183
		Gigantism	See 1183
	DIMINISHED SECRETION or HYPOPITUITARISM	Dystrophia Adiposogenitalis	See 1177
		Dwarfism	See 1176
Supra-renal Capsules	EXCESSIVE SECRETION	No definite disease, but general increased activity of bodily functions.	
	DIMINISHED SECRETION	Addison's Disease	

In addition to the above there is much evidence to show that Diabetes Mellitus may be due to disease of the Islands of Langerhans in the pancreas and that sudden death may result from an enlarged, persistent Thymus gland.

1124 CUTANEOUS AND SUB-CUTANEOUS TISSUE

- **1133 Atrophy**
  - The skin is unusually smooth and thin. The fingers become occurs quite frequently in nervous diseases, especially in
  - The hair falls out, either all over head, face and body (as in is not changed in appearance. Allied to this condition of severe pain, or psychic shock, or unknown cause (loss
  - Atrophy of the normal pigment of the skin; so that patches sons of dark complexion. The edge of the patch is more See also facial hemi-atrophy, 1179.
- **1134 Hypertrophy.**
  - The skin and mucous membranes everywhere appear thicke slightly, on pressure. The skin is sallow, dry and scaly. features are enlarged. Nails, teeth and hair break and f and hoarse. Response is slow and intellectuality very slu atrophied, or destroyed, by disease. The disease may follo and intestitial nephritis may be present. Is more common time of the climacteric. When it occurs in children they sence of the secretion of the thyroid gland and it can be roid gland.
  - The skin is thickened, generally or locally, infiltrated, very at their ends, and the fingers become much shortened and men and seems to be allied to myxedema. The disease of ends with an atrophy of the indurated patch (stadium at
- **1135 Eruptions.**
  - Clusters of vesicles filled with clear fluid, each cluster upon nerve roots and strictly limited to their distribution. Th accompanied, preceded and followed by severe pains in the for months after the rash has disappeared. Rarely, in se Lymphocytosis has been found in the cerebro-spinal fluid i
  - In some forms of nervous disease (especially in hysteria) el when the skin is irritated (urticaria scripta, dermograp do not. See also Angio-Neurotic Edema (1201).
  - Successive crops of bullae, which are at first small vesicles eral vesicles may coalesce. There may or may not be fe tense. A very fatal disease.
- **1136 Ulcerations.**
  - **With much loss of tissue.**
    - Ulcerations larger and smaller with sloughing and loss of phalanges and even whole fingers and toes. The whole process is painless and may in part be the result of traumatism in the analgesic parts.
      - No spa tribu tumo of le
      - Spastic to h tacti symp
    - Large, deep, sloughing ulcers commencing ally suffering from motor and sensory sure (sacrum, trochanters, etc.), espec
  - **With small loss of tissue. See also Raynaud's disease (1195).**
    - The ulceration usually commences on the extending deeper, until in many cases i ulcer very rarely occurs on the hand. I forms a sinus. Loss of knee-jerk, Ar ent in the majority of cases, while sug
    - Ulcerations more or less severe, the resul bronzed. Symptoms of neuritis (933) a

MPTOMS

PHIC DISORDERS

IS	DIAGNOSIS	
ssively curved and are striated. This change eral neurous are degenerated.	Glossy skin.	1160
ies, usually on the head and face. The skin hair in patches, or universally, in consequence	Alopecia (general or areata).	1161
hey are, of course, most noticeable in per- urrounding skin.	Vitiligo and Leucoderma.	1162
do not pit, or pit but e to cold. The body and re heavy. Voice is slow ed. The thyroid gland is gland. Arteriosclerosis d frequently occurs at the of the disease is the ab- dministration of the thy- — Occurring in adults.	Myxedema.	1163
Occurring in children.	Cretinism and Dwarfs (1092, 1177).	1164
of the phalanges become absorbed, especially disease is more common in women than in atch of edema (stadium oedematosum) and es are pigmented.	Scleroderma and Sclerodactyly.	1165
e clusters following the course of one or two sappears after a week or two. It is usually which it is situated. The pain may continue ry paralysis may be associated with herpes.	Herpes Zoster. Herpetic Ganglionitis or Neuritis. (940-78, 1235).	1166
appear, at times spontaneously, and always of urticaria sometimes itch and sometimes	Urticaria (1201).	1167
ear on the skin and mucous membranes. Sev- burning sensation and the pain may be in-	Pemphigus.	1168
disturbances are limited to the area of dis- All forms of sensibility are abolished. Small ve trunk, together with other manifestations	Leprous Neuritis.	1169
is usual, the trophic disturbances are limited temperature sense lost, with persistence of area. Kyphosis and spondylitis are common	Syringomyelia or Morvan's disease (553, 693, 840-2, 1009, 1152, 1187). (Figs. 25-7).	1170
d occurring only in bed-ridden patients usu- most always on parts subjected to much pres- t kept scrupulously clean.	Bed Sores. Decubitus.	1171
larger superficially, but slowly and painlessly foot and appears on its dorsum. Such an orn, which ulcerates and the pus, escaping, flexes and other symptoms of tabes are pres- a small minority.	Perforating Ulcer of Tabes and (rarely) Syringo-	1172
cases of arsenical neuritis, the skin is often	Neuritis (488-92, 824, 940-9, 1147).	1173

CHART XVIIc

Trophic Disorders of Fat, Bone and Joints

Comprising Numbers 1125 to 1128 and 1137 to 1141 on left side of Chart
and 1174 to 1188 on right margin

DIAGNOSTIC SYMPTOMS AND TESTS — ABSTRACT OF

1125 FATTY TISSUE.

1137 Atrophy.

One of the earliest symptoms of diabetes mellitus is an inability of the body excess of fat had been deposited. Patients lose weight and if the disease i urine shows the constant presence of sugar. Atrophy of fat and emaciati

1138 Hypertrophy.

Large and tender deposits of fat, in lumps or in layers, widely diffused over Arms and legs painful and tender, especially in the acute stage when the f frequently in middle aged women (often alcoholic or syphilitic).

Excessive accumulation of fat, especially on buttocks, pubes and breasts. Def

1126 BONE TISSUE.

1138a Failure in development.

Many cases occur, either congenitally or acquired in early life, in which the so that the individuals remain throughout life of abnormally small stature. due to atrophy or loss of function of the pituitary gland. Some of these inc (simple dwarfs or decidedly undersized men), while others show many p elsewhere, under infantilism and mongolism (1095), cretinism (1095, 1164 dro-dystrophia foetalis) there is a dystrophy of the epiphyseal cartilages, in in length; so that dwarfism results. The head is relatively long, the bridge their proximal segment, the hand is short, the fingers broad, of almost equal pelvis contracted, legs often bowed or knock-kneed and joints abnormally l Adults, as well as children, not infrequently become shorter in consequence as in rickets, osteitis deformans (1182), osteomalacia (1185), etc., and in

1139 Atrophy.

In cases of extensive acute anterior poliomyelitis and of cerebral palsy of chi growth or very slow growth of the part from disuse.

One side of the face is much smaller than the other, due to atrophy of all th and fat. The process is usually progressive. It seems to be caused by in geminal neuritis. Dryness, scaliness and loss of color of the skin are commo which a small area atrophies, which atrophy gradually extends laterally bones. The process continues until the entire half of the face is atrophied even to other parts of the body. One side of the tongue is usually atrophie accompanies the atrophy.

1140 Hypertrophy.

One side of the face is much larger than the other, due to enlargement of all ally progressive, and seems in some cases to be due to a periostitis.

The bones of the head and face are enlarged, diffusely or nodulated, and may them. Headache, neuralgia, blindness, deafness and facial paralysis are, t are not enlarged. Forehead is bulging and head is often of great size.

Disease commences late in life with slight pains, especially in legs. The bon jaw is not enlarged. The head enlarges, the legs and vertebral column bec patients become shorter (even as much as a foot or more) and their walk i

Symmetrical enlargement of all the tissues, but especially the bones of the ha etc. It comes on gradually, patient requiring larger and larger gloves and shouldered" (kyphosis). These changes are often associated with bitempo head and joints is a common symptom. The disease is caused by hypertro early life, before the epiphyses are joined by bone to shaft, gigantism inste

The hands and feet are enlarged, and the fingers and toes "clubbed." The bo shown by the X-ray. These symptoms are associated with chronic pulmona symptoms vary greatly in degree and extent; the mildest form being "clu

1141. Fragility.

In some persons the bones are unusually brittle and break and bend upon the bed. Some of these cases occur in old age (senility), others occur in mid lime salts (osteomalacia), while others occur in children. The disease cau fecta, osteopsathyrosis, etc. In many of these cases, the sclera show a bluis

1127 JOINT DISEASE.

Joints painless, enlarged, abnormally movable, especially hyperextension, cartilages eroded, effusion of synovial fluid, exostoses of bone. The exciting cause for these changes is often painless traumatism, at least in part.

- Joint involvement not uncommon. Usually in legs. — Knee-jerks ar Bladder sym
- Joint involvement rare. Usually in arms., — Knee-jerks are the lesion. painful and

1128 OTHER TROPHIC LESIONS.

Atrophy and Hypertrophy.

Atrophy or hypertrophy of different organs (mammary glands, tongue, etc.), frequently met with and may be due to disordered nervous action, but they value.

CHART XVIId

Ganglionic Disorders, Vaso-Motor Disorders

Comprising Numbers 1129, 1130 and 1142 to 1145 on left side of Chart
and 1191 to 1203 on right margin

1129 GANGLIONIC DISORDERS.

1142 Paralytic.

Ptosis of eyelid (due to paralysis of Müller's muscle), although patient c
of the levator palpebræ superioris. Contraction of pupil (myosis), v
briskly when eye is exposed to light and on convergence. Narrowing of
(exophthalmos). Intra-ocular tension diminished. The cilio-spinal refl
on the affected side of face, and also on side of neck, or of arm and thor
of cocaine in both eyes accentuates the symptoms by dilating the pupil
paralysed one.

1143 Irritative.

The symptoms are exactly opposite to those of paralysis of the cervical sy
widening of the palpebral fissure (Stellwag's sign) and delayed descent
sign). The Boston-Kocher's sign, an amplification of Graefe's sign, may

Exophthalmos, tachycardia, goitre, flushing, sweating, tremor, nervousness,
ward (Graefe's sign), widening of the palpebral fissure (Stellwag's sign
thyroid. The disease occurs much more frequently in women than in me
disorder of the cervical sympathetic ganglia, yet it is really due to excess
which are the reverse of those of myexdema (1163), can be produced b
be cured by extirpation of the thyroid.

Paroxysmal spasm or congestion of the bronchioles, often reflex from nasal
nervous temperament of most asthmatics, together with the very rapid o
may be due to a disturbance of the thoracic sympathetic. The paroxysm
prolonged expiratory murmur, make the diagnosis easy. Asthma is asso
be in part voluntary, in part reflex; also is usually associated with bron

1130 VASO-MOTOR DISORDERS.

1144 Vascular.

Paroxysmal attacks of subjective and objective coldness and pallor ("dead
or tip of nose or of ears or of all together. These attacks may last a few
lowed by an attack, in which the same parts become dusky blue, or purp
This is associated with pain. This attack may pass off, after several hou
tion of them, may become gangrenous and finally slough off. The necro
The disease is usually symmetrical. It is more common in cold weather
by working with hands. Paroxysmal hemoglobinuria and evidence of co
tacks. A hemiplegia and in other cases a coma, both transitory, have bee

Analogous to Raynaud's disease is gangrene of extremities occurring in mar
old age; either without the local syncope or local asphyxia, or with only

Paroxysmal attacks of formication, tingling, numbness and other paresthesia
tervals and exclusively in women. They seem to be brought on by overw
during the attack the skin becomes pale and blue. Similar symptoms so

Paroxysms of severe pain in one foot, rarely in both, rarely in hands and
increased by allowing foot to hang down, or by motion of it, or by cold.
redness and swelling of the whole, or part, of the sole of foot. Usually
to a simple vaso-motor neurosis. The neuritis, when present, is often as

Occurs in middle-aged or elderly persons and is associated with arterial dis
short walk and increases so that walking becomes impossible. It passes
ing the attack the feet are cold and there is absent or greatly diminished
Syphilis, alcohol and tobacco and injury seem to be common causes of th
grene of the feet. The arms are rarely involved. The disease seems to
motor spasm.

In many diseases, if lines or writing be traced on the skin with a sharp p
change to lines of bright redness, which persist for minutes or hours.

1145 Exudative or Secretory.

Paroxysmal attacks of localized edema of subcutaneous or submucous tiss
ing a few hours or days. The extent of the edema varies greatly. It may
extremity, or even more. It may cause death when occurring in the laryn
sure. They occur in hysteria and are usually associated with a neurasth
are associated with symptoms of digestive disorder, they are called urtic
eases except the itching. The disease often shows a strong heredity and n

Edema of the legs, unilateral becoming bilateral, bad heredity. The edema
being a sudden demarcation at the level of the joint. The edema may b

Some cases present paroxysmally or constantly a profuse sweating, usuall

F SYMPTOMS

DISORDERS

IPTOMS	DIAGNOSIS	
eyelid perfectly by an effort of will by contraction es not dilate when shaded, although it contracts ral fissure with retraction and lowering of eyeball) is abolished, flushing of skin and absence of sweat the third rib. In this condition, the instillation ising the eyelid in the healthy eye, but not in the	Paralysis of Cervical Sympathetic. Horner's Syndrome (455).	1191
tic. Dilation of pupil (mydriasis), exophthalmos, er eyelid when eye is turned downward (Graefe's in this disease and in exophthalmic goitre (1193).	Irritation of Cervical Sympathetic.	1192
l descent of upper eyelid when eye is turned down- and systolic murmur in vessels of neck and over although many of its symptoms may be referred to rtion of the thyroid gland. Many of its symptoms, dministration of thyroid gland, and the disease can	Exophthalmic Goitre (672).	1193
e. Freedom from symptoms in the interval. The l cessation of the attack, indicates that the disease ks of dyspnoea, with the abundant dry rales and with strong contraction of the diaphragm, which may	Asthma (616).	1194
" "local syncope") and tingling of fingers or toes es or hours and then may pass off, or may be fol- ek ("local asphyxia or cyanosis") from congestion. h abundant sweating, or the parts, or a small por- aot usually involve the whole of the cyanotic area. often brought on by putting hands in cold water, or n of other internal organs may occur in some at- bed in a few cases of this disease.	Raynaud's Disease. Symmetrical Gangrene (1011).	1195
bers of a family at varying ages from childhood to ndications of these conditions in some of the cases.	Family Gangrene.	1196
gers and hands. The attacks occur at irregular in- d by having the hands in cold water. In some cases s occur in the early stages of acromegaly (1183).	Acroparesthesia.	1197
ely in face, lasting a few minutes or a few hours, in, except in the earliest attacks, is accompanied by men only, and is generally due to a neuritis, rarely with atheromatous arteries.	Erythromelalgia (1010).	1198
A painful cramp occurs in muscles of legs after a a short rest to return if walking is resumed. Dur- ion in the dorsalis pedis or posterior tibial artery. lition. The disease not infrequently precedes gan- ally due to arteriosclerosis associated with vaso-	Intermittent Limping or Claudication. Dysbasia Angiosclerotica (555).	1199
e lines appear for a few seconds white, but soon	Dermographia (326, 1167).	1200
sing localized swellings, either white or red, last- half inch in diameter, or may extend over an entire ese swellings are not tender and do not pit on pres- ndition. If the swellings are red in color, itch and No sharp line can be drawn between the two dis- seems to be malarial.	Angio-Neurotic Edema and Urticaria. (1167). Quincke's Disease.	1201
limited above by the ankle, knee or groin; there nted with pyrexia or gastric disturbance.	Milroy's or Meig's Disease. Trophedema.	1202
ned, sometimes general.	Hyperhidrosis. Excessive Sweating.	1203

CHART XVIII

Syphilis of the Nervous System

Comprising Numbers 1205 to 1217

DIAGNOSTIC ANALYSI
SYPHILIS OF THE NI

DIAGNOSTIC SYMPTOMS AND TESTS

1205
SYPHILIS OF THE NERVOUS SYSTEM.

History of personal, or hereditary, syphilis. Physical evidence of syphilis; such as Wassermann reaction, a chancre or its scar, induration, mucous patches, a syphilitic rash or its copper colored scars, hazy cornea, notched teeth, furrows about angle of mouth, saddle nose, ptosis, iritis, enlarged glands, periosteal nodes, etc. (108, 175 to 180).

- Syphilitic Nervous Diseases.

  Symptoms of syphilis of the nervous system are very variable from day to day, transitory and manifold. They consist of paresis, rather than of complete paralysis. They usually show rapid improvement under K. I. and Hg. or Salvarsan. Nocturnal headache is common, as are also the Argyll-Robertson's pupillary reflex, unequal pupils and optic neuritis.

  - Cerebral symptoms.

    Although these symptoms can be divided into several, more or less well defined, groups, yet a combination of several or all of the lesions, in varying intensity, is not infrequent; so that a combination of the symptoms of several or all of the groups may be present in one case. Pure, uncomplicated cases of each type are, however, commonly met with.

    - Little or no lymphoc cerebro-spinal fluid f bar puncture.
    - Globulin and decided ocytosis is found in spinal fluid from puncture.

  - Spinal symptoms.

    (Both forms of spinal syphilis may occur together).

    - No globulin and littl lymphocytosis found bro-spinal fluid from puncture.
    - Globulin and decided ocytosis found in spinal fluid from puncture.

  - Cerebral and spinal symptoms. — Globulin and decided ocytosis in cere fluid.
  - Local peripheral symptoms. — Wassermann reaction blood. Normal cere fluid.

- Post-, or Meta-, syphilitic nervous disease. A term used very infrequently of late.

  - Cerebral symptoms.
  - Spinal symptoms.

  — Wassermann reaction blood, usually. Increased lymphocytosi bro-spinal fluid.

MPTOMS

SYSTEM

ABSTRACT OF SYMPTOMS		DIAGNOSIS	
Symptoms of cerebral tumor (507, 536). Other syphilitic symptoms may be present. Rapid course with irregular remissions and intermissions. The symptoms of cerebral compression are much less pronounced than in non-syphilitic tumors. Very amenable to anti-syphilitic treatment.		Isolated Cerebral Gumma.	1206
Symptoms of cerebral thrombosis (506). The attacks occur rather early in adult life. There are many prodromata. Nocturnal headache is common. The paralysis is moderate in degree, variable in intensity and often temporary. Mental derangements, often in the form of trance-like states, frequently occur. Branches of the basilar artery are involved most frequently, and the attack often occurs during sleep, or without coma during the day.		Cerebral Syphilitic Endarteritis and Thrombosis.	1207
Symptoms of meningitis (590, 605), which may be very slight and very variable. With severe headache (nocturnal) there may be some nausea and vomiting. Little or no elevation of temperature or retraction of neck. No tuberculin reaction or evidence of tuberculosis. This disease is rare in children.	Symptoms of cortical irritation (Jacksonian epilepsy, local headache and tenderness) and paralysis of cortical functions (aphasia, monoplegia, etc.). Mental derangement is common, and often takes the form of paresis (pseudo-paresis), but is amenable to anti-syphilitic treatment.	Syphilitic Meningitis of Convexity of Brain.	1208
	No symptoms of cortical irritation or paralysis of cortical functions. Paralysis of cranial nerves (especially the oculo-motorius), progressive, of irregular distribution and in varying degree. Drowsiness and stupor are common.	Syphilitic Meningitis of Base of Brain, including Kahler's Disease (Multiple Myeloma).	1209
Symptoms of Brown-Séquard's paralysis, or later of paraplegia (432, 509, 844, 975-81).		Isolated Spinal Gumma.	1210
Symptoms of myelomalacia (485, 513-4, 517-8, 550-1).		Spinal Syphilitic Endarteritis and Thrombosis.	1211
Symptoms of lateral sclerosis (525). (Fig. 26.)		Erb's Syphilitic Lateral Sclerosis.	1212
Symptoms of spinal meningitis, or of pachymeningitis (551, 605, 974, 1005). Rigidity of back. Girdle pains and radiating pains, exaggerated reflexes in legs. Some of these cases present the symptoms of progressive spinal muscular atrophy (548).		Syphilitic Meningitis of Cord and of Nerve Roots. (Meningo-myelitis, Pachymeningitis Cervicalis Hypertrophica.)	1213
A combination of the above symptoms, noted under 1208-9-13, in very varying extent and intensity. A clinical picture comprising cerebral and spinal symptoms and presenting great variations, which are impossible to describe in a few words.		Cerebro-Spinal Syphilis.	1214
Symptoms of neuritis (488-92, 824-5, 940-8).		Syphilitic Neuritis.	1215
Symptoms of general paresis (1106).		Paresis.	1216
Symptoms of locomotor ataxia (661).		Locomotor Ataxia. Tabes. (Fig. 27.)	1217

CHART XIX

Abnormal Cerebro-Spinal Fluid

Comprising Numbers 1220 to 1244

DIAGNOSTIC ANALYSIS

ABNORMAL CEREBI

TESTS AND DIA

- **1220 ABNORMAL CEREBRO-SPINAL FLUID.**
  - 1221 Globulin test positive. White cells increased.
    - 1223 Leucocytosis.
      - Weichselbaum's diplococcus intra-cellularis meningitidis or rarely Pneumococcus. — Fluid may l Tension i
      - Weichselbaum's diplococcus, Pneumococcus, Pfeiffer's bacillus, Streptococcus, Staphylococcus, Typhoid bacillus or Bacterium coli, etc. — Fluid usuall high tensi
      - Tubercle bacillus. — Fluid usual cate coa high tens
    - 1224 Lymphocytosis.
      - Tubercle bacillus.
      - Wassermann and Colloidal Gold reactions positive. — Fluid clea bacteria.
      - Wassermann and Colloidal Gold reactions negative. — Tension is but not v
  - 1222 Globulin test negative. White cells not increased.
    - 1225 No lymphocytosis. No leucocytosis.
      - No bacteria and Wassermann negative. — Fluid clear sion.

PTOMS				
FLUID				
IGNS			DIAGNOSIS	
loudy. lly.	Occurs in epidemics.	Symptoms of epidemic Cerebro-spinal meningitis (591).	Epidemic Cerebro-spinal Meningitis.	1226
under	Occurs sporadically.	Symptoms of sporadic or purulent cerebro-spinal meningitis (592).	Sporadic Purulent Meningitis.	1227
deli- under	Acute course.	Symptoms of tuberculous meningitis (593).	Acute, or sub-acute Tuberculous Meningitis.	1228
	Chronic course.		Chronic Tuberculous Meningitis.	1229
from	Tremor and mental symptoms.	Symptoms of Paresis (1106).	Paresis.	1230
	Ataxia.	Symptoms of Tabes (661).	Tabes.	1231
	Symptoms not typically characteristic of paresis or tabes, being due to a cerebro-spinal meningitis.		Cerebro-spinal Syphilis (1208-9,	1232
	Motor paralysis.	Symptoms of acute anterior poliomyelitis (495). (Figs. 26-7).	Acute Anterior Poliomyelitis.	1233
		Symptoms of Encephalitis Lethargica (1047).	Encephalitis Lethargica.	1234
	Herpetic rash.	Symptoms of herpes zoster (1166).	Herpes Zoster.	1235
reased	Epidemic. High fever.	Symptoms of Typhus.	Typhus Fever.	1236
	Choked disc usually present.	Symptoms of cerebral or spinal tumor (507, 578, 587).	Tumor.	1237
	Choked disc may be present.	Symptoms of cerebral or spinal abscess (508, 578, 587).	Abscess.	1238
		Symptoms of hydrocephalus (405, 905, 961).	Hydrocephalus.	1239
	Pain and spasm in back.	Symptoms of decided spinal irritation, with slight or no paralysis (524).	Hematorrhachis.	1240
l ten-	Headache.	Symptoms of serous meningitis (594).	Serous Meningitis.	1241
	Apoplexy.	Symptoms of cerebral or spinal hemorrhage (503, 524, 1063-4).	Hemorrhage.	1242
	Albumen and casts.	Examination of the urine shows albumen and casts. Edema, headache, dyspnoea, etc., usually present.	Uremia.	1243
	Anemia.	Examination shows anemia, pallor, etc., or acute infections, or some similar conditions.	Anemia.	1244

PART III

Localization

OF

Lesions Within the Nervous System

BY

A CONSIDERATION OF THE
PARALYTIC AND IRRITATIVE SYMPTOMS
RESULTING FROM THEM

CHART XX

Spinal Localization

A. According to altitude
Comprising Numbers 1250 to 1267

B. According to situation in transverse area
Comprising Numbers 1268 to 1279.

A—TABLE OF SYMPTOMS IN TRANSVERSE LESIONS AT DIFFERENT LEVELS OF THE CORD

Modified from Wichman

Involved	Motor Conditions			Reflex Conditions		Sensory Conditions
	Paralysis	Paresis	Actions lost or impaired	Absent	Increased in partial lesions	Anesthesia with a zone of hyperesthesia surrounding it or limiting it above
1250 V Sacral	None.	Coccygeus.	Elevation of coccyx.	Anal.	None.	Skin over sacrum and anus.
1251 IV Sacral	Coccygeus.	Levator ani. Sphincter ani. Detrusor urinae. Transversus perinei. Erector penis.	Elevation of coccyx. Elevation of anus Sphincter ani. Ejection of urine. Vaginal constriction. Compressor urethrae.	Erection of penis diminished.	None.	Slightly larger area than above extending over inner portion of gluteal region.
1252 III Sacral	Sphincter ani. Levator ani. Detrusor urinae. Transversus perinaei. Erector penis Compressor urethrae.	Rectum.	Defecation disturbed. Retention of urine later followed by dribbling. Ejaculation lost. Erection possible but paretic.	Ejaculation lost. Erection diminished. Tendo-Achillis	None	As above, and perineum, genitals and upper part of inner surface of thighs. (Testicle sensitive to pressure).
1253 II Sacral	Sphincter ani. Levator ani. Detrusor urinae. and other muscles as in 3d sacral.	Pyriformis. Obturator internus. Gemellus superior. Gluteus maximus. Biceps femoris. Gastrocnemius. Soleus. Tibialis posticus. All the small muscles of foot.	Outward rotation of thigh. Retraction of thigh. Flexion of knee. Plantar flexion of foot. Standing on the toes. Raising inner margin of foot. Defecation and Retention of urine as in 3d sacral.	Ejaculation. Erection. Plantar weakened.	None.	As above, and the posterior surface and outer surface of thighs.

TABLE OF SYMPTOMS IN TRANSVERSE LESIONS AT DIFFERENT LEVELS OF THE CORD (Continued)

Modified from Wichman

Segment Involved	Motor Conditions			Reflex Conditions		Sensory Conditions
	Paralysis	Paresis	Actions lost or impaired	Absent	Increased in partial lesions	Anesthesia with a zone of hyperesthesia surrounding it or limiting it above
1254 I Sacral	Muscles of anus. Muscles of bladder. Muscles of genitals. Pyriformis. Abductor hallucis. Flexor hallucis brevis. I-IV dorsal interossei. I-III plantar interossei. III-IV lumbricales. Abductor minimi digiti. Opponens minimi digiti.	Gluteus maximus. Obturator internus. Gemellus superior. Gluteus medius. Gluteus minimus. Biceps femoris. Semimembranosus. Semitendinosus. Popliteus. Gastrocnemius. Soleus. Tibialis posticus. Peroneus longus. Peroneus brevis. Flexors of toes. Extensors of toes.	Retention of feces. Retention of urine or dribbling. Erection and ejaculation impossible. Outward rotation of thigh impaired. Internal rotation impaired. Flexion of knee difficult. Plantar flexion of foot. Raising inner margin of foot. Raising outer margin and dorsal flexion of foot. Flexion and extension of toes, adduction of great toe, abduction of little toe, etc.	Plantar weakened. Achilles-tendon reflex. Ejaculation. Erection. Micturition. Defecation. Gluteal.	None	As above, and a strip on posterior and outer surface of lower legs and of dorsum of foot and especially of toes.
1255 V Lumbar	Muscles of anus and rectum. Muscles of bladder. Muscles of genitals. Pyriformis. Biceps femoris. Flexors of toes. Peroneus longus. Peroneus brevis.	Gemellus superior. Gemellus inferior. Gluteus medius. Gluteus minimus. Semimembranosus. Semitendinosus. Gluteus maximus. Tensor fasciae femoris. Gastrocnemius. Soleus. Extensors of toes. Tibialis anticus.	Defecation. Micturition delayed, dribbling. Erection and ejaculation impossible. Outward rotation of thigh very difficult. Inward rotation impaired. Flexion of knee difficult. Retraction of thigh very difficult. Flexion of foot barely possible. Flexion of toes impossible. Extension of toes weak, except great toe, which may be dorsally flexed. Raising inner margin of foot difficult. Raising outer margin of foot impossible.	Ejaculation. Erection. Micturition. Defecation. Gluteal.	Plantar. Tendo-Achillis.	As above, and back of thighs and legs and inner and outer margin and sole of feet.

TABLE OF SYMPTOMS IN TRANSVERSE LESIONS AT DIFFERENT LEVELS OF THE CORD (Continued)

Modified from Wichman

Segment Involved	Motor Conditions			Reflex Conditions		Sensory Conditions
	Paralysis	Paresis	Actions lost or impaired	Absent	Increased in partial lesions	Anesthesia with a zone of hyperesthesia surrounding it or limiting it above
1256 IV Lumbar	Muscles of rectum and anus. Muscles of bladder. Muscles of genitals. Obturator internus. Pyriformis. Gemelli. Gluteus medius. Gluteus minimus. Gluteus maximus. Biceps femoris. Semimembranosus. Semitendinosus. Popliteus. Gastrocnemius. Soleus. Flexors of toes. Extensors of toes. Peroneus brevis. Peroneus longus. Tibialis anticus.	Obturator internus. Rectus femoris. Vastus externus. Vastus internus. Adductor magnus. Adductor brevis. Adductor minimus. Gracilis.	Defecation, with fecal incontinence. Micturition, with dribbling. Erection and ejaculation impossible. Outward rotation of thigh weak. Inward rotation impossible. Retraction of thigh impossible. Flexion of knee lost. Plantar flexion of foot lost. Flexion and extension of toes lost. Raising outer margin of foot. Raising inner margin. Extension of thigh weak. Adduction difficult.	Patellar may be wanting.	Plantar.	As above, and inner side of lower legs and dorsum of feet and strip on outer posterior surface of thighs.
1257 III Lumbar	Muscles of anus, bladder and genitals. Outward rotators of thigh. Inward rotators of thigh. Retractor of (flexor) thigh. Flexors of knee. Plantar flexors of foot. Flexors of toes. Extensors of foot. Vastus externus.	Vastus internus. Rectus femoris. Crureus. Adductors of thigh. Flexors of thigh at the hips.	All movements of legs are lost, except that extension of legs is barely possible and that the thigh can be flexed on body by the psoas and iliacus. Defecation and micturition are destroyed. Urine and feces dribble and cannot be retained.	Patellar and cremasteric.	Ankle-clonus may exist.	As above, and whole of legs except a triangular area on front of thigh with base at Poupart's ligament.

TABLE OF SYMPTOMS IN TRANSVERSE LESIONS AT DIFFERENT LEVELS OF THE CORD (Continued)

Modified from Wichman

Involved Segment	Motor Conditions			Reflex Conditions		Sensory Conditions
	Paralysis	Paresis	Actions lost or impaired	Absent	Increased in partial lesions	Anesthesia with a zone of hyperesthesia surrounding it or limiting it above
1258 II Lumbar	Paralysis of all muscles of lower extremity, except psoas.	Psoas.	Complete paralysis of legs, rectum and bladder. As above.	Patellar, Achilles and cremasteric.	Achilles may be increased. Plantar.	Whole of legs and pelvis. (Testicles not sensitive to pressure.)
1259 I Lumbar	Total paralysis of whole lower extremity, psoas included.		As above.	Cremasteric and Achilles.	Patellar absent or increased.	As above, and groins and front of scrotum and penis.
1260 XII to III Dorsal	Paralysis of lower extremity, and gluteal region. Paralysis of abdominal and dorsal regions, gradually added as the site of the lesion ascends.		As above, and paralysis of muscles of respiration causes diaphragmatic breathing and dyspnoea.	Epigastric and umbilical reflex.	Patellar, cremasteric, Achilles and plantar.	As above, and a band running around body about two segments below the one involved and limited above by a narrow zone of hyperesthesia.
1261 II Dorsal	As in 3d dorsal.		As above.	All below lost in complete division of cord.	All subjacent reflexes.	As above, and a strip on the inner side of the upper arms.
1262 I Dorsal	All muscles of trunk and lower extremities.	Flexion of fingers. Muscles of the little finger. III and IV interossei. Lumbricales. Pronator quadratus. Lower part of pectoralis major. Lower part of pectoralis minor.	As above and weakness in flexion of fingers. Pronation disturbed.	Oculo-pupillary symptoms. All below lost in complete division of cord.	All subjacent reflexes.	As above, and a strip on the inner side of the forearms.

TABLE OF SYMPTOMS IN TRANSVERSE LESIONS AT DIFFERENT LEVELS OF THE CORD (Continued)

Modified from Wichman

Segment Involved	Motor Conditions			Reflex Conditions		Sensory Conditions
	Paralysis	Paresis	Actions lost or impaired	Absent	Increased in partial lesions	Anesthesia with a zone of hyperesthesia surrounding it or limiting it above
1263 VIII Cervical	Paralysis of muscles of trunk and lower extremities. Abductor of little finger. Adductor of thumb. Flexor of the little finger. Opponens minimi digti III and IV interossei. Lumbricales.	Flexors of the little finger. Opponens minimi digiti. Flexor subl. digitorum. Flexor profun. digitorum. Flexor carpi ulnaris. Extensors of the thumb and fingers. Triceps (slight). Latissimus dorsi (lower part). Pectoralis major. Pectoralis minor. Scalenus medialis. Scalenus posticus.	As above. Hand weak. Extension of arm. Int. rotation and retraction of arm. Adduction of arm.	Oculo-pupillary symptoms. All below lost in complete division of cord.	All below.	As above, and the fingers, except volar surface of the thumb and the ulnar surface of the little finger. The cervical sensory nerve roots supply the same area of the skin in common, especially in the hands and fingers. Hence the anesthesia is slight and uncertain.
1264 VII Cervical	Lower extremities and trunk. Flexor profundus digitorum (ulnar side). Flexor carpi ulnaris. Small hand muscles. Pronator quadratus.	Extensors, Flexors and Abductors of thumb. Extensor indicis. Extensors of the fingers (movements barely possible). Supinator longus. Biceps (very slightly paretic.) Triceps Pectoralis major. Serratus magnus (slight). Latissimus dorsi. Teres major.	As above and Hand very weak. Retraction and inward rotation of arm. (Winged scapulae)	Arm reflexes. Forearm reflexes. Palmar reflex. All below lost in complete cord division.	All below.	As above, and most of the hands and a small strip on the anterior, another on the posterior, surface of the forearm.

TABLE OF SYMPTOMS IN TRANSVERSE LESIONS AT DIFFERENT LEVELS OF THE CORD (Concluded)

Modified from Wichman

	Sensory Conditions	Motor Conditions			Reflex Conditions		Conditions Sensory
	Paralysis	Paresis	Actions lost or impaired	Absent	Increased in partial lesions		Anesthesia with a zone of hyperesthesia surrounding it or limiting it above.
1265 VI Cervical	Muscles of lower extremity and trunk. Muscles of fingers (including thumb) and hand. Triceps. Pectoralis major. Latissimus dorsi. Teres major. Infraspinatus. Serratus magnus.	Coraco-brachialis. Biceps. Brachialis anticus. Supinator brevis. Deltoid. Scaleni. Splenii. Deep head and neck muscles.	As above and movements of fingers and thumb impossible. Extension of forearm. Flexion of forearm weak. Supination very weak. Adduction of arm and inward rotation. Adduction, retraction and external rotation. (Winged scapulae.) Raising of arm. Rotation of head. Fatal in a few days or weeks.	Arm reflexes. Extensor forearm reflexes. All below lost in complete cord division.	All below.		As above, and whole of hands and fingers and radial side of forearm.
1266 V Cervical	Muscles of lower extremities and trunk. All the muscles of the arm, forearm, hand and fingers; even the deltoid, coraco-brachialis and brachialis anticus. Deep cervical muscles. Intercostals.	Levator anguli scapulae. Scaleni. Diaphragm (because of filaments from V cervical segment to phrenic nerve), or spread of injury from 5th to 4th cervical segment. Trapezius and sterno-cleido-mastoid are intact.	As above and shoulders raised with difficulty. Rotation and flexion of head. Dyspnoea. Fatal in a few hours or days.	Scapular and tendon reflexes of paralysed muscles in arms. All below lost in complete cord division.	All below.		As above, and whole of arms, except tip of shoulder.

1267 IV-I Cervical — Total cross-lesions from the fourth cervical segment upward are rapidly fatal, because of complete paralysis of the diaphragm and intercostals.

Total cross-lesions of the brain-stem are rapidly fatal for the same reason.

B.—TABLE OF SYMPTOMS ACCORDING TO AREA INVOLVED IN TRANSVERSE SECTION OF THE SPINAL CORD

1268 Lesions of anterior horns. { Acute anterior poliomyelitis (495, 789, 1148, 1233, 1324.) / Chronic atrophic paralysis (547-8, 695, 1150, 1324.) }

1269 Lesions of posterior horns; sensory disturbances (1322.)

1270 Lesions of pyramidal tract; Spastic Paraplegia (525-47, 800, 1212, 1384-6-9, 1406.)

1271 Lesions of posterior columns; Tabes (661, 785-6, 1322, 1360, 1363-4, 1406.)

1272 Lesions of direct cerebellar tracts; Hemiataxia (653.)

1273 Lesions of postero-lateral columns; Ataxic Paraplegia (526, 660, 799, 1360, 1406.)

1274 Lesions of spino-thalamic tract; Dissociation of sensation (812, 1369.)

1275 Lesions of anterior gray commissure; Syringomyelia (552, 693, 798, 840-2, 1370-2.)

1276 Lesions of entire lateral half; Brown-Sequard paralysis (442, 509, 844, 975-82.)

1277 Lesions of posterior spinal ganglion; Herpes Zoster (940, 978, 1166, 1235.)

1278 Lesions of posterior nerve roots; Tabes and Herpes Zoster.

1279 Lesions of anterior nerve roots; Atrophic paralysis.

CHART XXI

Cerebral Localization

Comprising Numbers 1290 to 1309

CHART XXIa

Cerebral Localization in the Medulla and Pons· Ganglia at Base

Comprising Numbers 1290 and 1292

VARIX OF SYMPTOMS IN

LOCALIZATION

Seat of Lesion		Palsies of Motion	Palsies of Sensation	Action Impa
[illegible] Lesion involving anterior half of the Medulla Oblongata. Avellis' syndrome.		[illegible] hemiplegia [illegible]. Homolateral half of tongue. Dysphagia and [illegible] homolateral arm and leg. In some cases arm and leg may be paralysed on both sides, but not equally so. Occasionally only leg on one side and arm on the other are paralysed.	[illegible] a posterior part of [illegible] half of tongue. All forms of sensation in junction and throughout the [illegible] half. Analgesia and thermic anæsthesia of homolateral half of face and homolateral half of body. Anæsthesia of one side, or of both sides of the body.	Articulation, p [illegible] diction, res [illegible] action [illegible] us [illegible] and of arms [illegible] one or both s
Bulbar and Nuclear Bulbar Syndrome.				
[illegible] of the small transverse area of the [illegible] (Fig. [illegible])				
[illegible] Posterior Inferior Cerebellar [illegible] (Fig. [illegible])		None.	Analgesia, thermic anæsthesia and tingling in homolateral side of face, tongue and nasal, and in contralateral arm and leg.	Deglutition, arti
Lesion in the Pons Varolii. (Fig. [illegible])	Confined to the tegmentum.	Crossed paralysis: hemiplegia [illegible]. Muscles of expression of homolateral half of face and the external recti [illegible] and contralateral arm, leg and half of tongue. Millard-Gubler's syndrome—[illegible].	None, unless [illegible] from pressure and then [illegible] hemiparesis.	Articulation, wi [illegible] Sitation. Mo homolateral h and of contra and leg.
	Confined to the tegmentum.	Muscles of expression of homolateral half of face and of external recti. Foville's paralysis—[illegible]. Homolateral external recti and arm and leg may be slightly involved.	[illegible] hemianalgesia and thermic anæsthesia and at times hemianæsthesia. Anæsthesia and especially analgesia of homolateral half of face. Hemianæsthesia [illegible]. Very rarely deafness. Rarely [illegible] of sensation.	Articulation, whistling. Mo homolateral ha
Lesion in Middle and Upper Segment of Pons.	Confined to the tegmentum.	Complete contralateral hemiplegia.	Usually all forms of sensation in homolateral half of face. Occasionally also hemianæsthesia of contralateral half of body.	Chewing and usu [illegible] tion. Movem [illegible] lateral half
	Confined to the tegmentum.	Complete lesion of [illegible] on the side of the lesion. May be complete hemiplegia of slight degree from pressure.	Palsies of all forms of sensation on homolateral half of face. Contralateral hemianalgesia. May be contralateral hemianæsthesia.	Chewing and usu [illegible] tion. Conjug [illegible] ment of eyeb the same sid [illegible] lesion.

SE LESIONS OF BRAIN-STEM

ILLA AND PONS

	Reflexes Altered	Vertigo	Ataxia	Muscle Sense	Secretory and Other Disturbances
s- :- g, e n	Tendon reflexes increased with Babinski and ankle-clonus on opposite side. Cutaneous reflexes may or may not be increased.	Usually present.	Usually present and of both motor and cerebellar type. Homolateral.	Usually lost, especially if motor ataxia be present.	Miosis and pseudo-ptosis (ophthalmoplegia sympathica) and salivation are common. Cheyne-Stokes's respiration (425).
	Normal except in certain cranial nerves.	Usually present at onset.	Extreme in homolateral arm and leg.	Lost in homolateral arm and leg.	Increased secretion of sweat in contralateral side. Tendency to fall towards the side of the lesion. Nystagmus frequent.
s- of e. m	Tendon reflexes increased with Babinski and ankle-clonus on opposite side. Cutaneous reflexes may or may not be increased.	Often present.	No motor, but there may be cerebellar, ataxia.	Normal.	Conjunctivitis is frequent in eye of same side. May be a tendency to fall or to turn to one side. Salivation.
n, of	Normal or slightly exaggerated as above.	Usually present.	Usually present on the same side as the lesion.	Lost on the same side as the lesion.	Conjunctivitis is frequent in the eye of the same side. Salivation.
r- r-	Tendon reflexes increased with Babinski and ankle-clonus on the opposite side. Cutaneous reflexes may or may not be increased.	Often present.	No motor, may be cerebellar, ataxia.	Normal.	Ulceration of cornea may occur. May be a tendency to fall or turn to one side
r- e- d e	Normal or may be slightly exaggerated.	Present.	May be motor and cerebellar ataxia.	Lost on the same side as the lesion.	Ulceration of the cornea may rarely occur. A slow rhythmic tremor of the arm and leg of opposite side may be present.

CHART XXIb

Cerebral Localization: Ganglia at Base

TABLE OF SYMPTOMS IN TRANSVERSE LESIONS OF BRAIN STEM AND CEREBELLUM

EAT OF LESION		PARALYSIS OF MOTION	PARALYSIS OF SENSATION	ACTIONS LOST OR IMPAIRED	ALTERED REFLEXES	VERTIGO	ATAXIA	MUSCLE SENSE	SECRETORY AND OTHER DISTURBANCES
a bri: bral incles.	Lesion confined to the pes or foot.	Some, or all, of the ocular muscles (except external rectus) on the same side, combined with a contralateral hemiplegia, usually complete. Hemiplegia alternans oculomotoria. (Weber's syndrome, 430).	None. Contralateral hemianesthesia, or hemianalgesia and thermic hemianesthesia, or both. Deafness may be present, if lesion be bilateral.	Movement of eyeball. Use of contralateral half of the body.	Tendon reflexes increased, with Babinski and ankle-clonus, on opposite side. Cutaneous reflexes may or may not be increased.	Usually absent.	None.	Normal.	Tremor resembling that of paralysis agitans of contralateral arm and leg (Benedikt's syndrome). (431) A slow, rhythmic tremor of arm and leg of opposite side may be present.
	Lesion confined to the tegmentum.	One or more ocular muscles, except the abducens.		Movement of eyeball.	Tendon reflexes normal.	Present.	Cerebellar type.	Impaired.	
a bri: ora dri- ina. s. 19.)	Lesion confined to anterior pair (nates).	Bilateral, more or less extensive, of all ocular muscles, except the abducens.	May be blindness without choked disc or other lesion.	Movement of eyeball.	Pupil reflex lost to both light and accommodation.	Usually absent.	May be absent.	Normal.	Nystagmus (at times vertical), squint, pupils often unequal.
	Lesion confined to posterior pair (testes).	None or may be slight paralysis as above, or of trochlearis.	May be deafness, if lesion be bilateral.	None, except chewing at times.	Normal.	Usually present.	Present. Of cerebellar type.	Normal.	May be slow, rhythmic tremor of arm and leg of opposite side, especially on voluntary motion.
im.		None.	None.	Walking and standing	Normal or slightly exaggerated. Rarely abolished.	Usually present.	Cerebellar ataxia with hypotonia almost always present.	Normal.	Nystagmus (80), tendency to fall to one side, occipital headache is frequent, cerebellar fits may occur.
dle r incles.		None.	None.	Walking, standing and sitting.	Normal or slightly exaggerated.	Usually present.	Usually present with hypotonia of the cerebellar type.	Normal.	Tendency to fall or to turn eyes, head or body to one side. Rotatory movements, more or less pronounced, choreic-spasms in homolateral half of body, and vertical divergence of the eyeballs sometimes occur.

Lesion of inferior cerebellar peduncles cause lateropulsion; those of the superior cerebellar peduncles cause choreiform movements and cerebellar ataxia.

Fractures, tumors, etc., at base of skull may cause many of the above symptoms according to their position, but their early and characteristic symptom is paralysis of one or more of the cranial nerves. Symptoms of paralysis predominate over those of irritation.

of ium. 19.) Small lesions, not so extensive as to involve the entire lateral half of the brain stem, may occur at any point. The symptoms of these lesions depend upon the function (physiology) of the part affected and will naturally vary greatly. The location of such a lesion in a transverse section will depend upon what longitudinal fiber tracts are involved, and in longitudinal section upon what cranial nuclei and nerve tracts are involved, as shown by the symptoms present in any case. A study of the figures at the end of this book is essential for the localization of such lesions and will serve this purpose better than a long verbal description.

CHART XXIc

Cerebral Localization: Ganglia at Base

LOCALIZING SYMPTOMS IN LESIONS OF GANGLIA AT BASE OF BRAIN

Seat of Lesion	Diagnostic Symptoms
1298 Optic Thalamus. (Fig. 17) (837a)	Is the highest and most important center for complicated automatic actions. It is the seat of the emotion of anger and probably, in large part of pleasure and well being. Disease of this organ may give rise to few characteristic symptoms. There may be hemianopia (pulvinar and external geniculate involvement) with hemiopic pupillary reaction and contralateral sensory disturbances with consequent incoordination. In lesions of the optic thalamus occasionally a slight irritation of the skin is not felt at all, while a stronger one is felt inordinately. Absence of emotional expression in face, even when not paralysed. Vaso-motor disturbances may occur in opposite side of body. Isolated analgesia and thermic anesthesia do not occur in lesions above the optic thamus.
1299 Corpus Striatum. (Fig. 17) — Nucleus Lenticularis and Nucleus Caudatus.	In rare cases a lesion of the nucleus lenticularis may be of such a form as to injure the anterior and posterior part of the posterior limb of the internal capsule, while its middle part escapes. In such cases there results a hemiplegia which involves the leg and face more than the arm. Dysarthria is a not uncommon symptom and in some cases the symptoms of sensory irritation and incoordination described under lesions of the optic thalamus have been present. When the ganglia on both sides are affected, voluntary voiding of urine may be impossible, while automatic involuntary voiding may occur at regular intervals. The most characteristic symptoms due to lesions in the corpus striatum are rigidity (Parkinson's disease—677), choreiform movements (Huntington's chorea—624) and athetosis (Post-hemiplegic—632 and Bilateral—631). These symptoms also form part of the syndromes known as: pseudo-sclerosis—667, progressive lenticular degeneration and dystonia lenticularis (668).
Internal Capsule.	Lesions in the anterior limb of the internal capsule cause either no symptoms or a paralysis of contralateral half of face. There may be ataxia and athetoid movements.
	Lesions in the anterior two-thirds of the posterior limb of the internal capsule cause a total contralateral hemiplegia of the body. This hemiplegia consists purely of a muscular paralysis and never produces a paralysis of the cortical functions such as aphasia, alexia, etc; but may produce dysarthria.
	Lesions in the posterior third of the posterior limb of the internal capsule cause hemianesthesia and loss of muscle sense on the opposite side of the body.
	Lesions at the extreme posterior end of the posterior limb of the internal capsule, in addition to hemianesthesia, cause contralateral hemianopia, deafness, only if the lesion be bilateral, and often the symptoms of motor irritation, described under lesions of optic thalamus.
1300 Corpus Callosum.	No diagnostic symptoms. Symptoms of ingravescent character, gradual development of hemiplegia, with slight hemiplegia of the other side also, drowsiness, dysarthria and anarthria. Death in coma (Bristow's syndrone).
1301 Island of Riel, Claustrum and External capsule. (Fig. 17)	Lesions in this area produce disturbances of speech, grouped under the general term paraphasia, and may produce anarthria, the result of complete aphasia.
1302 Pituitary Gland.	Hypertrophy, tumor, hemorrhage and some other lesions of the gland associated with excess of secretion may cause acromegaly or gigantism, in addition to a progressive bi-temporal hemianopia, terminating in blindness. A defect or atrophy of the gland associated with a diminution of secretion in early life may cause dwarfism and may produce pituitary eunuchismus or adiposogenital degeneration with excess of fat and a defect in the formation of the genitals. In any case of pituitary disease there may be polyuria, polydipsia and occasionally glycosuria and very rarely an escape of cerebro-spinal fluid from the nose (hydrorrhoea nasalis). In some cases of pituitary disease there are no symptoms.
1303 Pineal Gland.	Abnormal growth of hair and deposition of fat. Abnormalities of genitals (at times with attacks of sexual excitement, eunuchismus). Excessive growth in height of body (dyspinelismus). In consequence of involvement of adjacent tissue, bilateral ocular paralysis, nystagmus, pupil abnormalities, ataxia, and perhaps disturbances of hearing may be present.

CHART XXId

Cerebral Localization: Lobes of Brain

LOCALIZING SYMPTOMS IN LESIONS OF CEREBRAL HEMISPHERES

Seat of Lesion		Diagnostic Symptoms
1304 FRONTAL LOBE Contains the centers for all the skilled acts, especially the left lobe. Large lesions in the frontal lobes may cause a change in character and disposition of the patient. Many lesions, especially tumors, cause Jacksonian epilepsy, especially when situated in posterior part of lobe; while lesions in anterior part of lobe may cause epileptiform convulsions. Ataxia sometimes occurs in tumors in the frontal lobe. (Fig. 15)	The ascending frontal convolution. Lesions in this region may cause awkwardness (cortical ataxia or apraxia) rather than paralysis.	Lesions in the upper fourth of this convolution may cause Jacksonian epilepsy commencing in, and motor paralysis of, the contralateral leg. Very large lesions (hemorrhage, tumors, etc.) in this region may cause also paralysis of the homolateral leg in a lesser degree.
		Lesions in the middle half of the convolution may cause Jacksonian epilepsy commencing in, and awkwardness of or loss of skill or complete paralysis of the contralateral arm, Very minute lesions in the upper part of this region may affect only the shoulder; in the lower part, only the hand.
		Lesions in the lower fourth of this convolution may cause Jacksonian epilepsy commencing in, and paralysis of, the contralateral half of face and neck. Very minute lesions in the upper part of this region may affect only the eyes; in the lower and anterior part, the tongue and larynx.
	The base of the middle left frontal convolution.	Small lesions in this area may cause in right-handed persons, agraphia, and in many cases Jacksonian epilepsy, commencing in the contralateral arm.
	The base of the inferior left frontal convolution.	Small lesions in this area may cause, in right-handed persons, motor aphasia, and in many cases Jacksonian epilepsy, commencing in the right side of the face.
1305 PARIETAL LOBE Contains the centers for cutaneous and muscular sensation. Many lesions, especially tumor, cause Jacksonian epilepsy when situated in the anterior portion of this lobe; while lesions in posterior portion may cause epileptiform convulsions. (Fig. 15)	The ascending parietal convolution.	Lesions in the upper fourth of this convolution may cause some blunting of cutaneous sensibility, and especially astereognosis in contralateral leg and foot.
		Lesions in the middle half of this convolution may cause some blunting of cutaneous sensibility, and especially astereognosis in contralateral arm and hand.
		Lesions in the middle half of this convolution may cause some blunting of cutaneous sensibility. and especially astereognosis in contralateral half of face.
	The left angular gyrus.	Lesions in this region may cause loss of muscular sense and motor ataxia in the contralateral arm and leg.
	The rest of the parietal cortex.	Deep lesions in this region in right-handed persons may cause alexia and hemianopia.
1306 TEMPORAL LOBE Contains, on the left side, the centers of scnsory speeeh. Lesions may cause epileptiform convulsions. (Fig. 15)		Lesions in the posterior portion of the left superior temporal convolution in right-handed persons, mav cause sensory aphasia (psychic deafness.)

LOCALIZING SYMPTOMS IN LESIONS OF CEREBRAL HEMISPHERES
(Concluded)

Seat of Lesion		Diagnostic Symptoms
1307 OCCIPITAL LOBE Contains the centers of sight. Lesions may cause epileptiform convulsions. (Fig. 15)	Neighborhood of calcarine fissure.	Lesions in this area cause contralateral homonymous hemianopia. A lesion limited to the superior lip of this fissure causes quadrantic hemianopia or tetartanopia of the contralateral lower quadrants of field of vision. A lesion limited to the inferior lip of this fissure causes loss of contralateral upper quadrants of the field of vision.
	Rest of occipital lobe.	Lesions in this area may cause loss of power of recognition of persons and things (psychic blindness).
1308 Cortical Lesions. (Fig. 15)		Many lesions cause a mixutre of paralysis and convulsions over a limited area which in some cases may slowly grow larger. The intelligence of the patient is always more or less impaired.
1309 Sub-Cortical Lesions.		Localized lesions in the white substance of the brain (centrum ovale) may involve the fibers of the corona radiata. Such lesions when lying close to the cortex will cause the symptoms characteristic of lesions of the overlying cortex, but are not quite so sharply defined. In especial, Jacksonian epilepsy and mental symptoms are less pnonounced than when resulting from cortical lesions.

CHART XXII

Cerebro-Spinal Localization

TOPICAL DIAGNOSIS

LOCALIZATION OF LESIONS FROM ANALYSIS OF SYMPTOMS

1310
PARALYSIS
The most important of all localizing symptoms.

- **1312**
  The reflexes in the paralysed area are abolished (except in 1310 and 1329) A lesion of the peripheral neurons.
  - **1314** Sensation alone, in all its forms is lost or impaired
  - **1315** Motion alone is lost or impaired.
  - **1316** Both motion and sensation are lost or impaired

  } See Chart XXII a.
  - **1317** Special forms of peripheral paralyses. — See Chart XXII b.
  - **1318** Sensory paralysis dominant. Little or no motor paralysis. — See Chart XXII c.
- **1313**
  The reflexes are present (except in 1357 and 1359) A lesion of the central neurons.
  - **1319** Motor paralysis dominant. Little or no sensory paralysis. — See Chart XXII d.
  - **1320** Both motor and sensory paralysis well marked.

} See Chart XXII e.

1311
Jacksonian Epilepsy, together with other symptoms of cerebral disease.

For diseases and lesions accompanied by *motor paralysis* see 469, by *motor spasm* see 570, by *ataxia* see 638, by *tremor* see 639, by *nystagmus* see 640, by *fibrillation* see 641, by *local paralysis* see 636, by *local spasm* see 637, by *disorders of speech* see 735, by *disorders of gait* see 736, by *anesthesia and analgesia* see 811-15, by *disorders of special senses* 808-10, by *pain* see 931, by *vertigo* see 932, by *mental disorders* see 1036, *by trophic disorders* see 1121, by *vaso-motor disorders* see 1130, by *ganglionic disorders* see 1129, by *syphilis* see 1205, by *abnormal cerebro-spinal fluid* see 1220.

CHART XXIIa

Cerebro-Spinal Localization Paralysis with Abolished Reflexes

TOPICAL DIAGNOSIS

LOCALIZATION OF LESION FROM ANALYSIS OF SYMPTOMS

		Diagnostic Symptoms and Tests			Localization	
Reflexes Abolished	1314 Sensation alone, in all its forms, is lost or impaired.	Area of anesthesia, etc., lies within the area of distribution of one or more nerves.	Onset acute or sub-acute.	Nerve involved, if palpable, is tender on pressure. No symptom of disease of central organs usually, unless nuclei are affected.	Lesion is in one or more sensory cranial nerves or nuclei or sensory end-organ; the nerve affected depending upon its anatomical distribution (822). (Figs. 19-21, 33, 38).	1321
		Area of anesthesia, etc., lies within the area of distribution of one or more nerve roots.	Onset acute or chronic.	Nerves involved, if palpable, are not tender. May be symptoms of disease of central organs.	Lesion is in corresponding sensory nucleus in the brain stem, or in the posterior horn of spinal cord, or in column of Burdach, or in posterior nerve root. (Figs. 19-21, 24-6).	1322
	1315 Motion alone is lost or impaired.	The paralysis is limited to muscles supplied by one or more nerves. (Figs. 19-21.)	Onset acute or sub-acute. No fever at onset.	Nerve involved, if palpable, is tender on pressure. No symptoms of disease of central organs. All the muscles supplied by the nerve are paralysed, usually.	Lesion is in one or more motor cranial nerves, or a mild lesion of mixed spinal nerves; the nerve affected is the nerve supplying the paralysed muscles (489-93). (Figs. 19-21, 33, 38).	1323
		The paralysis is limited to muscles supplied by one or more nerve roots. (Figs. 19-21)	Onset acute or chronic. May be fever at onset.	Nerve involved, if palpable, not tender. May be symptoms of disease of central organs. Often only a portion of the muscles innervated by the nucleus are paralysed.	Lesion is in corresponding motor nucleus within brain stem, or in anterior horn of spinal cord, or in the anterior nerve root (493-5). (Figs. 19-21, 24-6).	1324
	1316 Both motion and sensation are lost or impaired.	Unilateral: Motor and sensory paralysis is within the area of distribution of one spinal nerve.	Onset acute or sub-acute. No fever at onset.	Nerve involved; tender on pressure. No symptoms of disease of central organs.	Lesion in one spinal nerve (489). (Figs. 33, 38).	1325
		Unilateral: Motor or sensory paralysis is within the area of distribution of several nerves from one plexus.			Lesion in brachial or lumbar plexus (490). (Figs. 32, 38).	1326
		Bilateral: Motor and sensory paralysis extends over legs or arms or both, or even more generally.	Onset acute or sub-acute. May be fever at onset.	Nerves involved tender on pressure. No symptoms of disease of central organs.	Muscles show weakness, tenderness and rapid atrophy. Lesion of many spinal and (rarely) cranial nerves also (multiple neuritis) (488).	1327
				Nerves involved not tender. There are disturbances of organic reflexes and other symptoms of organic disease of central organs.	Legs alone are paralysed and exhibit trophic disturbances. Anesthesia of rectum and bladder.	Great pain. May be deformity of lumbar spines. Symptoms less symmetrical and bed-sores less common than in lumbar lesions. Domain of anterior crural nerve may be normal when lesion is low. Lesion of cauda equina (487). (Fig. 29). 1328
						Little pain. May be deformity of lower dorsal spines. Symptoms symmetrical. Bed-sores always present. No portion of legs escapes. Lesion of lumbar enlargement of spinal cord (484-7). (Fig. 24-6). 1329
					Both legs and arms are paralyzed. There are trophic disturbances in arms but not in legs. Reflexes are abolished in arms, exaggerated in legs. (549-52). Lesion of cervical enlargement of spinal cord (Fig. 24-6). 1330	

CHART XXIIb

Cerebro-Spinal Localization

Comprising Numbers 1317 and 1331 to 1333 on left side of chart
and 1334 to 1352 on right margin

T

LOCALIZATION OF LE

PERIPHERAL PARA

DIAGNOST

- 1317 SPECIAL FORMS OF PERIPHERAL PARALYSIS. REFLEXES ABOLISHED IN PARALYSED AREA, EXCEPT IN 1345.
  - 1331 DISTURBANCES OF VISION. (808).
    - Blindness of entire field of vision of one ey light.
    - Bitemporal hemianopia is present. The out present. Acromegaly or other symptoms
    - Nasal hemianopia is present. The inner hr is present. May be symptoms of a lesion
    - Homonymous hemianopia is present. Iden pupillary reflex is present, i. e., reflex is ab toms of a lesion in the middle fossa of th
  - 1332 PARALYSIS OF OCULAR MUSCLES (700).
    - All muscles of one eye paralysed. Eyeball
    - All muscles supplied by third cranial nerve are paralysed at once.
      - No hemip
      - Paralysis
      - Tremor o motion.
    - Partial or progressive paralysis of muscles
    - Paralysis of external rectus muscle.
      - No hemipl
      - Hemiplegi power o nerve m
  - 1333 FACIAL PARALYSIS (703).
    - Lower branch of facial only, or mainly, paralysed.
      - Other sym eration
    - Both lower and upper branches of facial nerve equally paralysed.
      - Paralysis c
      - No hemiple tory and
      - Associated
      - No deafnes Low not taste. A
      - Hyperakusi
      - No hyperak
      - No hyperak

IS OF SYMPTOMS

HED REFLEXES

TESTS	LOCALIZATION	
e is atrophied. Pupil does not respond to	Lesion in optic nerve (897-8).	1334
ision is blind. Hemiopic pupillary reflex is turcica (1279) may be found.	Lesion is in the central part of optic chiasm (362, 817, 864, 894).	1335
ie eye is blind. Hemiopic pupillary reflex ie cranium	Lesion is in outer margin of optic chiasm (362, 817, 865).	1336
of each field of vision are blind. Hemiopic of retina is excited by light. Other symp-	Lesion is in the optic tract or external geniculate body of opposite side (862-95).	1337
ce of disease within orbit.	Lesion is within the orbit (915).	1338
es paralysed.	Lesion of 3rd cranial nerve trunk or nucleus (700). (Fig. 18).	1339
side.	Lesion involving one crus cerebri (676).	1340
side present at rest and exaggerated on	Lesion of red nucleus or rubro-spinal tract on same side as motor oculi paralysis (431, 676).	1341
nerve (700).	Lesion of 3rd cranial nucleus, in whole or in part (700). (Fig. 18).	1342
s paralysed, especially the facial.	Lesion of 6th cranial nerve or nucleus (1346-7). (Figs. 19, 20).	1343
emianesthesia of opposite side. Loss of yes to right or left. Facial or auditory	Diffuse lesion of Pons Varolii (538, 885). (Figs. 19, 20).	1344
ain present. Electrical reaction of degen- resent.	Lesion above nucleus of facial nerve in cerebral hemispheres or in crura cerebri. (Figs. 15, 19).	1345
side. Often abducens paralysis.	Lesion in Pons Varolii. (Figs. 19, 20).	1346
ly. Other cranial nerves, especially audi- .	Lesion of nucleus of facial nerve. (Figs. 19, 20).	1347
id vertigo without disease of the ear.	Lesion of facial nerve trunk at base of brain (Fig. 19).	1348
nitus aurium, due to stapedius paralysis. s also, are painful to hear. No loss of n of tears.	Lesion of nerve above geniculate ganglion (928). (Fig. 36).	1349
r two-thirds of tongue of same side.	Lesion of facial nerve between geniculate ganglion and stapedius branch. (Fig. 36).	1350
rior two-thirds of tongue of same side.	Lesion of facial nerve between stapedius and chorda tympani branches. (Fig. 36).	1351
nderness near stylo-mastoid foramen.	Lesion of facial nerve below chorda tympani branch. (Fig. 36).	1352

CHART XXIIc

Cerebro-Spinal Localization

Comprising Numbers 1318 and 1353 to 1359 on left side of Chart
and 1360 to 1383 on right margin

TO

LOCALIZATION OF LES

ANESTHESIA WIT

DIAGNOSTIC SYMPTOMS AND TESTS

- 1318 SENSORY PARALYSIS DOMINANT. LITTLE OR NO MOTOR PARALYSIS. TENDON REFLEXES PRESENT OR EXAGGERATED.
  - 1353 ANESTHESIA with or without ANALGESIA.
    - Limited to one or both legs.
      - Marked ataxia. — Anesthesia mar of muscle se
      - Slight ataxia. — Anesthesia slig be cerebral s
    - Limited to one arm. — Slight ataxia. — Anesthesia sli other cerebr paralysis.
    - In both arms and both legs. — Marked ataxia. — May be other in arms and
    - In arm and leg of same side.
      - Marked ataxia. — May be other in arm and
      - Slight ataxia. — Anesthesia sli May be oth
    - In arm and leg of one side and in other side of face. — Moderate ataxia. — May be paraly of the eyeb
    - In arm, leg and face of same side. — Slight ataxia.
      - No Jacksonian
      - Jacksonian ep
  - 1354 ANALGESIA with THERMIC ANESTHESIA, but little or no tactile anesthesia, is present. DISSOCIATION OF SENSATION.
    - In one or both legs.
      - Usually unilateral. — No trophic d ataxia.
      - Usually bilateral. — Trophic distu usually abo
    - In one or both arms.
      - Usually unilateral. Leg of same side also involved. — No trophic di
      - Usually bilateral. Legs of normal sensibility. — Trophic distu especially i
    - In arms, or legs, or both.
      - Bilateral usually, marked ataxia. — May be othe paraplegia)
      - Unilateral, slight ataxia.
        - Hemianopia a
        - Jacksonian e thesia prese
        - Symptoms of
        - In contralater
  - 1355 HOMONYMOUS HEMIANOPIA. — Identical halves of each field of vision (right or left) are blind. No of the occipital lobes may be present.
  - 1356 HOMONYMOUS TETARTANOPIA, QUADRANT HEMIANOPIA. — Identical quadrants of each field of vision (right or left) are blind. anesthesia or other paralysis. May be other cerebral symptoms of
  - 1357 PSYCHIC BLINDNESS. — Patient is not blind, but cannot recognize things by sight, though he
  - 1358 SENSORY APHASIA.
    - Auditory. — Patient is not deaf, but cannot understand words spoken to him, alth memory for spoken words.
    - Visual. — Patient is not blind but cannot understand written words, although h memory for written words. Alexia.
  - 1359 ASTEREOGNOSIS. — Patient is not anesthetic, or very slightly so, but cannot recogniz e o sight.

	LOCALIZATION	
e other spinal symptoms, especially loss	Lesion in one or both posterior columns of cord in dorsal region. Same side if unilateral (654, 786). (Figs. 24-6).	1360
n foot. Almost always unilateral. May epilepsy, etc.	Lesion in upper one-fourth of posterior central convolution in contralateral cerebral cortex. (Fig. 15).	1361
hand, astereognosis marked. May be onian epilepsy). Usually some motor	Lesion in middle one-half of posterior central convolution in contralateral cerebral cortex. (Fig. 15).	1362
spnoea common. Loss of muscle sense	Lesion of posterior columns of cord in cervical region (654, 786). (Figs. 24-6).	1363
spnoea common. Loss of muscle sense	Lesion of posterior column of cord on same side, in cervical region (654, 786). (Figs. 24-6).	1364
hand and foot. Astereognosis marked. especially Jacksonian epilepsy.	Lesion in upper three-fourths of posterior central convolution of contralateral cerebral cortex. (Fig. 15).	1365
erves. Paralysis of conjugate deviation	Lesion in tegmentum of pons Varolii on same side as the facial anesthesia (885). (Fig. 20).	1366
ia common.	Lesion of posterior part of internal capsule of contralateral hemisphere (861, 1299). (Fig. 17).	1367
eminanopia. Mental deterioration.	Lesion of superior parietal lobule of contralateral hemisphere (657). (Fig. 15).	1368
urbance of organic reflexes. Usually	Lesion in periphery of opposite lateral column of cord in dorsal region (1372). (Figs. 24-6).	1369
ic reflexes disordered. Tendon reflexes lvanced cases. (Figs. 24-6).	Lesion in central gray matter (anterior commissure) of cord in lumbar enlargement. In central gliosis the lesion may extend upwards to the cervical enlargement and involve the arms secondarily (840-2, 1372).	1370
in without loss of muscle sense.	Lesion in periphery of the opposite, or of both, lateral columns of the cord in the cervical region (1373). (Figs. 24-6).	1371
don reflexes usually abolished in arms,	Lesion in central gray matter (anterior commissure) of the cord in cervical enlargement (Syringomyelia) (553, 693, 840-2, 1009, 1152-70-87, 1370). (Figs. 24-6).	1372
Always some motor paralysis (spastic	Lesion of lateral columns of cord (653, 1212, 1369, 1371, 1406). (Figs. 24-6).	1373
present. Other cerebral symptoms.	Lesion of posterior part of contralateral internal capsule (861). (Fig. 17).	1374
bral symptoms usually present. Anes-	Lesion of inferior parietal lobule of contralateral hemisphere (657). (Fig. 15).	1375
erior, Inferior Cerebellar Artery (1291).	Lesion (softening) of the lateral half of the pons (1291).	1376
afness.	Lesion of ponto-cerebellar angle on side of deafness (428).	1377
x. Other cerebral symptoms of lesions	Lesion of edges of calcarine fissure of occipital lobe, or of fasciculus of Gratiolet of contralateral cerebral hemisphere (362, 817, 890, 1307-19-21). (Fig. 16).	1378
reflex. No hemi- lobes. — Lower quadrant of field of vision.	Lesion of upper lip of contralateral calcarine fissure (363, 817, 1307-19-21).	1379
Upper quadrant of field of vision.	Lesion of lower lip of contralateral calcarine fissure (363, 817, 1307-19-21). (Fig. 16).	1380
g. He has forgotten what he has seen.	Lesion of cortex of occipital lobe of left cerebral hemisphere (232, 1307). (Fig. 15).	1381
em when he sees them written. Has no	Lesion of cortex or subcortex of posterior part of left superior temporal convolution or in the association fibers connecting the superior temporal with the inferior frontal convolution (222, 775). (Fig. 15).	1382
en he hears them spoken. He has no	Subcortical lesion of the Angular Gyrus (777, 1403).	1382a
touch, although he can by the sense of	Lesion in cortex or subcortex of the posterior central convolution of contralateral hemisphere (229, 354). (Fig. 15)	1383

CHART XXIId

Cerebro-Spinal Localization

Comprising Numbers 1319 on left side of chart
and 1384 to 1403 on right margin

TOPICAL
LOCALIZATION OF LESION F
MOTOR PARALYSIS WITH

	DIAGNOSTIC SYMPTOMS AND TESTS		
	Limited to one or both legs. Organic reflexes not disordered.	Symptoms bilateral usually.	May be other spinal symptoms. of sensation in legs.
		Symptoms unilateral usually.	May be other cerebral symptom lepsy.
	Limited to both arms and both legs. Organic reflexes not disordered.	No sensory paralysis. No cerebral symptoms. Often ataxia an arms and legs.	
		Usually some sensory paralysis. Dysarthria and dysphagia. Par ing with position of lesion.	
	Limited to one arm.	Occasionally some slight sensory paralysis. Jacksonian epilepsy common.	
	Limited to arm and leg of same side.	Dissociation of sensation and ataxia may be present. Organic cerebral symptoms.	
		Usually some sensory symptoms. Dysarthria and dysphagia cranial nerves frequent.	
		Usually some sensory symptoms. Jacksonian epilepsy and other	
1319 MOTOR PARALYSIS DOMINANT. LITTLE OR NO SENSORY PARALYSIS. TENDON REFLEXES PRESENT OR EXAGGERATED.	Limited to lower branch of facial nerve.		Jacksonian epilepsy and other common. Often complicated w
	Limited to arm and lower branch of facial nerve of same side.		
	Limited to arm and leg of same side and hypoglossus nerve of opposite side.		Usually some sensory symptoms Paralysis of some other crani abducens paralysis.
	Limited to arm and leg of same side and lower branch of facial nerve of opposite side.		
	Limited to arm and leg of same side and motor oculi nerve of opposite side.		Usually some sensory symptoms nerves common
	Limited to arm and leg and lower branch of facial nerve on same side.	Symptoms of paralysis rather than of irritation. Not progressive.	Usually other cerebral symptoms toms.
			Often sensory symptoms present. pression on opposite side of fac
		Symptoms of irritation. Jacksonian epilepsy.	No objective sensory symptoms.
	DYSARTHRIA and DYSPHAGIA	Paralysis of some of the cranial nerves and usually of arm and l	
	AGRAPHIA	Loss of power of writing, although arm is not paralysed.	
	MOTOR APHASIA	Loss of power of speaking some or all words. Limited vocabula muscles of speech not paralysed.	
	ALEXIA	Inability to read, although patient can see and can speak.	

IS OF SYMPTOMS

ED REFLEXES

	LOCALIZATION	
nd dissociation	Lesion of homolateral, or of both lateral, columns of cord in dorsal region (1369, 1371-3). (Figs. 25-7.)	1384
acksonian epi-	Lesion of upper part of anterior central convolution of contralateral hemisphere, cortical or subcortical (leg center). (Fig. 15).	1385
f sensation in	Lesion of lateral columns of the cord in the cervical region (525). (Figs. 25-7.)	1386
l nerves vary-	Lesion of the brain stem (involvement of pyramidal tract in the medulla, pons or crura cerebri). (Figs. 19-22.)	1387
bral symptoms	Lesion in cortex or subcortex of middle one-half of anterior central convolution of contralateral hemisphere (arm center). (Fig. 15.)	1388
isordered. No	Lesion of contralateral lateral column of cord in cervical region. (Figs. 25-7.)	1389
lysis of some	Lesion in the brain stem (involving the pyramidal tract). (Figs. 19-22.)	1390
rtical disease.	Lesion in cortex or subcortex of upper three-fourths of anterior central convolution of contralateral hemisphere. (Fig. 15.)	1391
rtical disease ia.	Lesion in cortex or subcortex of inferior part of anterior central convolution of contralateral hemisphere (face center). (Fig. 15.)	1392
	Lesion of cortex or subcortex of lower three-fourths of anterior central convolution of contralateral hemisphere (arm and face centers). (Fig. 15.)	1393
nd dysphagia. ion, especially	Lesion of medulla on same side as the hypoglossus paralysis (rare condition). (Fig. 21.)	1394
	Lesion in bridge portion of pons on same side as the facial paralysis. (Fig. 20.)	1395
other cranial	Lesion in pes cruris cerebri on same side as the motor oculi paralysis. (Fig. 19.)	1396
sensory symp-	Lesion in anterior part of posterior limb of internal capsule of opposite hemisphere. (Fig. 17.)	1397
emotional ex-	Lesion in posterior part of optic thalamus and corpus striatum of opposite hemisphere. (Fig. 17.)	1398
hasia.	Lesion throughout anterior central convolution of contralateral hemisphere (cortex or subcortex). (Fig. 15.)	1399
	Lesion in tegmentum of pons or medulla (284-5.) (Figs. 20-1.)	1400
	Cortical or subcortical lesion at base of middle frontal convolution of left cerebral hemisphere in right handed person (227-8, 779). (Fig. 15.)	1401
be made and	Cortical or subcortical lesion at base of inferior left frontal convolution in right handed person (221, 774). (Fig. 15.)	1402
	Subcortical lesion of left angular convolution in right handed person or involving the association fibers connecting the inferior frontal convolution with the occipital lobe in the left cerebral hemisphere (228, 777). (Fig. 15.)	1403

CHART XXIIe

Cerebro-Spinal Localization

TOPICAL DIAGNOSIS

LOCALIZATION OF LESION FROM ANALYSIS OF SYMPTOMS

MOTOR AND SENSORY PARALYSIS WITH EXAGGERATED REFLEXES

Diagnostic Symptoms and Tests			Localization	
1320 Both motor and sensory paralysis well marked. Reflexes present or exaggerated, except in 1406.	Limited to both legs.	Paralysis severe. No ataxia. Organic reflexes much disordered. Some of the trunk reflexes are lost. Vertical extent of lesion is shown by the absence of the different trunk reflexes. Upper limit of lesion shown by the zone of hyperesthesia, including the anesthesia above.	Transverse lesion of spinal cord in dorsal region. (Myelitis). (516-9, 829.)	1404
		Motor paralysis and exaggerated reflexes in one leg; anesthesia, analgesia, and thermic anesthesia in the other leg.	Unilateral lesion of the cord. Brown-Sequard's Paralysis. (432.)	1405
		Paralysis not so extreme. Marked ataxia. Loss of muscle sense. Organic reflexes not at all, or slightly, disordered. Trunk reflexes not abolished. Knee-jerks and other leg reflexes may be increased or abolished.	Lesion both in lateral and posterior columns of cord. (Ataxic Paraplegia). (526, 660, 799). (Figs. 25-7.)	1406
	Limited to both arms and both legs.	No involvement of cranial nerves. Priapism. Dyspnoea. Very dangerous, usually fatal.	Transverse lesion of spinal cord in cervical region. (512-5, 830). (Figs. 25-6.)	1407
		Involvement of some cranial nerves. Dysarthria and dysphagia. Very dangerous, usually fatal.	Lesions on both sides of brain stem (medulla, pons or crura cerebri, according to cranial nerves involved). (Figs. 19-21.)	1408

JACKSONIAN EPILEPSY

1311 JACKSONIAN EPILEPSY	Spasmodic twitching of head and eyes to one side. Twitching may remain limited to these muscles or may extend to other muscles of face and neck and arm and later to leg of same side or may finally extend to muscles of both sides of body.	Lesion in or near base of middle frontal convolution of contralateral hemisphere. (Fig. 15.)	1409
	Spasmodic twitching commences in one side of face. Twitching may remain limited to these muscles or may extend to others as above.	Lesion in or near lower quarter of the central convolutions of contralateral hemisphere. (Fig. 15.)	1410
	Spasmodic twitching in hand or arm. Twitching may remain limited to these muscles or may extend to face or to leg or to both simultaneously of same side and may later extend to muscles of other side of body also.	Lesion in or near middle half of the central convolutions of contralateral hemisphere. (Fig. 15.)	1411
	Spasmodic twitching of foot or leg. Twitching may remain limited to these muscles, or may extend to arm and later to face of same side and still later to muscles of the other side of body. (Figs. 15, 16.)	Lesion in or near upper quarter of central convolutions or paracentral lobule of opposite hemisphere.	1412
	Spasmodic twitching, commencing simultaneously, in arm and face of same side, which later extends to muscles of the leg of the same side and still later to muscles of the opposite side of the body.	Lesion near and equally distant from motor area of face and arm in contralateral hemisphere. (Fig. 15.)	1413
	Spasmodic twitching commencing in arm and leg of same side, which may later extend to face of same side and may later extend to muscles of the other side of body.	Lesion near and equally distant from motor area of arm and leg in contralateral hemisphere. (Fig. 15.)	1414
	Spasmodic twitching commencing in face and arm and leg of same side, which may later extend to muscles of opposite side.	Lesion in inferior parietal lobule of contralateral hemisphere. (Fig. 15.)	1415

PLATES

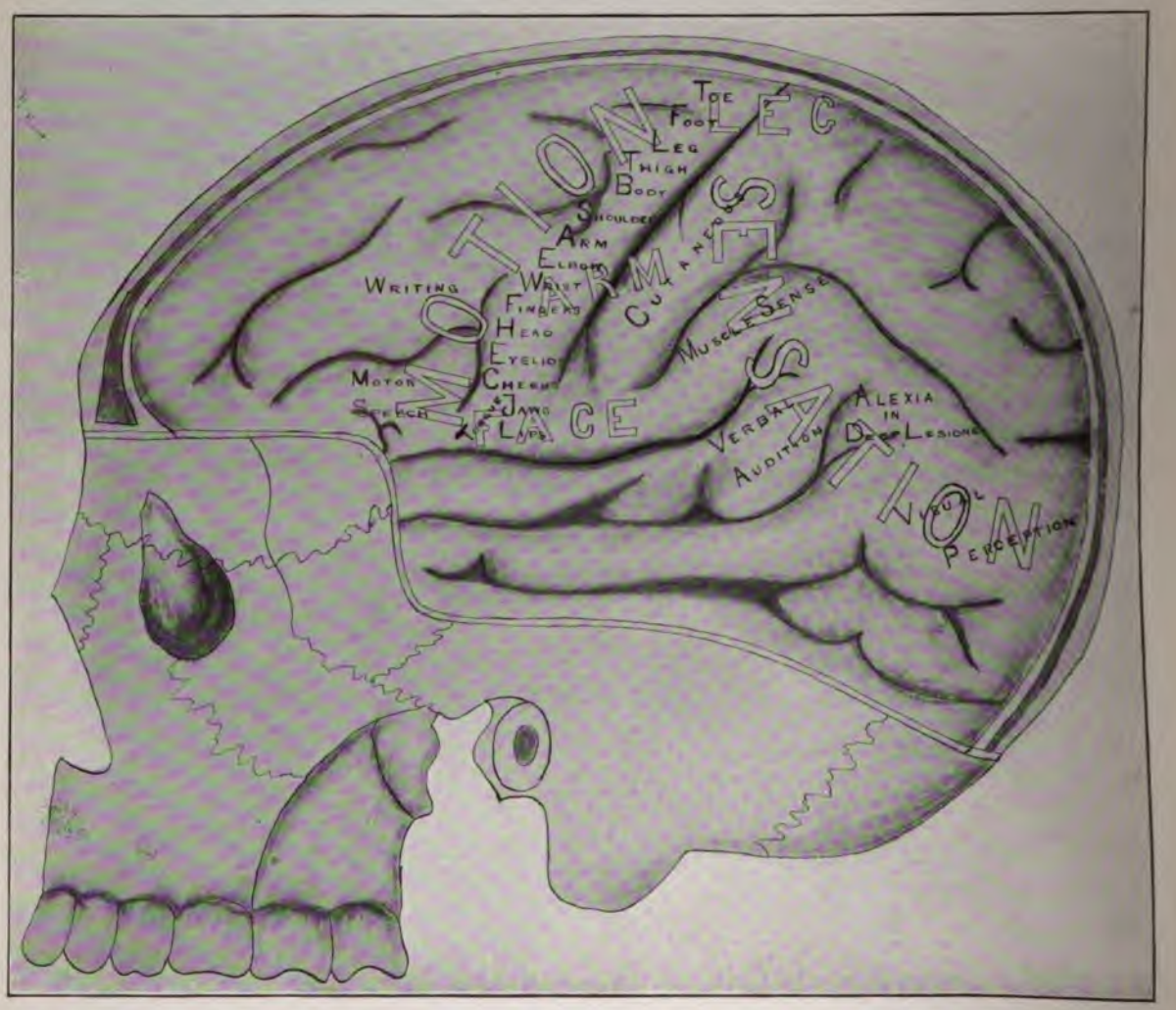


FIG. 15

Schematic representation of the convex surface of the left cerebral hemisphere, showing the motor and sensory areas, and the location of the cortical functions.

See 1304-9--61-2-5-8-72-81-3-5-8-91-2-3-9, 1401-3-10-5.

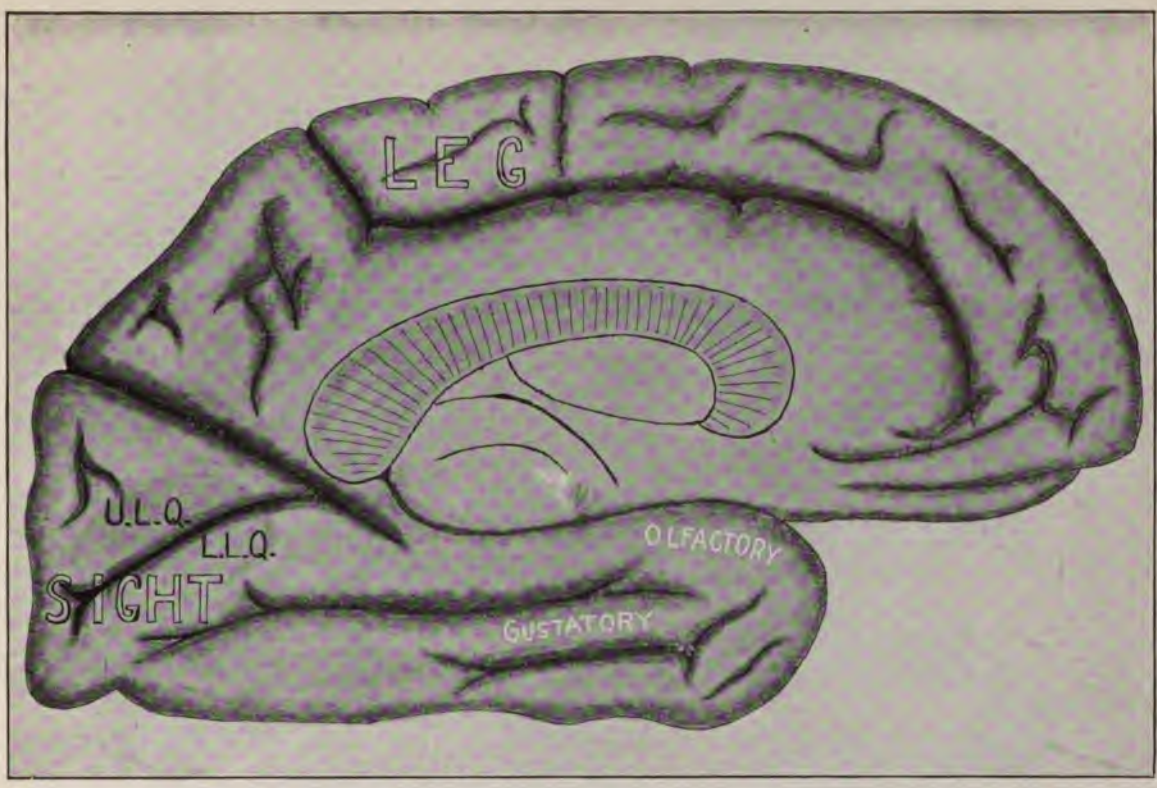


FIG. 16

Schematic representation of the median surface of the left cerebral hemisphere. U. L. Q. = Upper left quadrant of both retinae. L. L. Q. = Lower left quadrant of both retinae.

See 856-7-60, 1307-78-80, 1412.

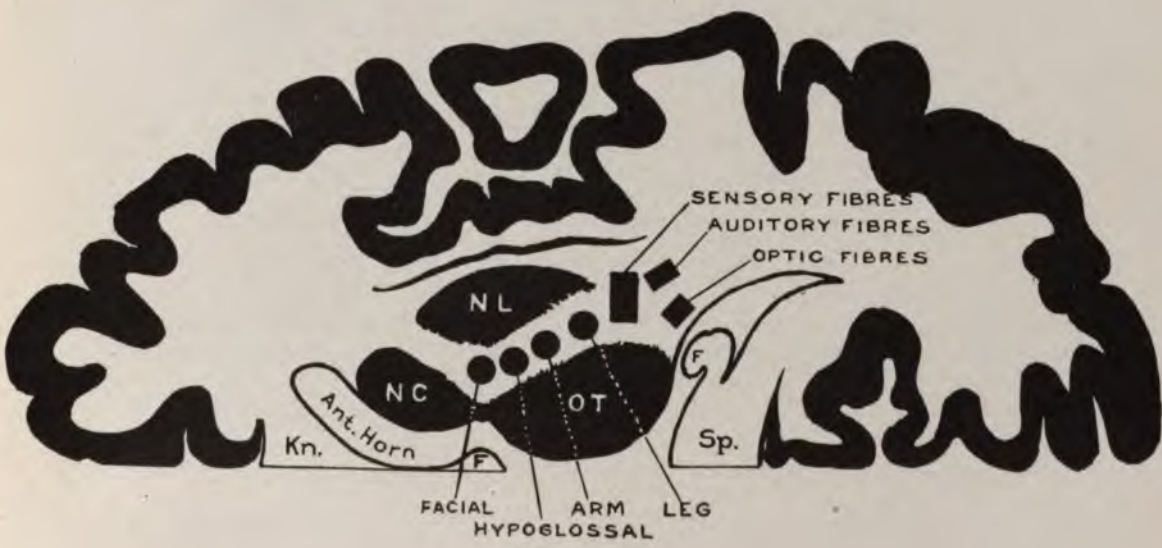


FIG. 17

Horizontal Section through Right Hemisphere showing the principal tracts situated in the Internal Capsule; Kn, Genu of Corpus Callosum; F, Fornix; NC, Caudate Nucleus; NL, Lenticular Nucleus; OT, Optic Thalamus, Sp, Splenium of Corpus Callosum.

See 1298-9, 1367-74-97-8.

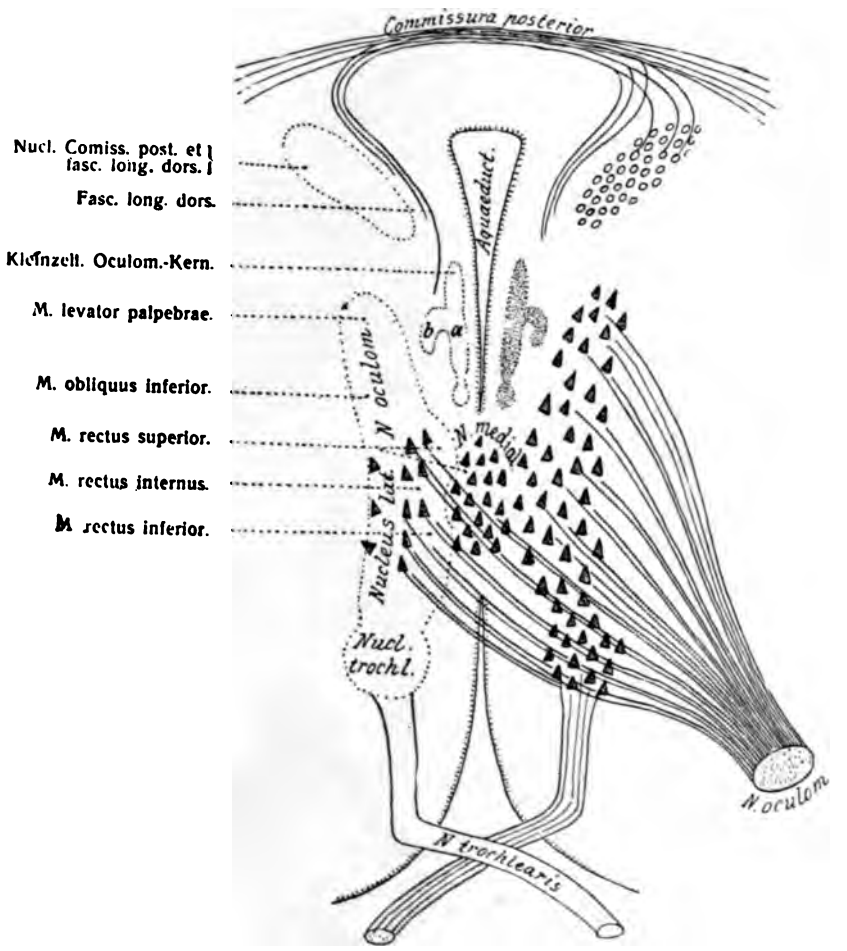


FIG. 18

Schematic representation of the nuclei situated beneath the floor of the Sylvian aqueduct, showing the origin of the posterior commissure, the oculo-motor and trochlearis nerves, as well as the nuclear localization of the centers for the individual ocular muscles (after Edinger.)

See 692, 700, 818, 1332.

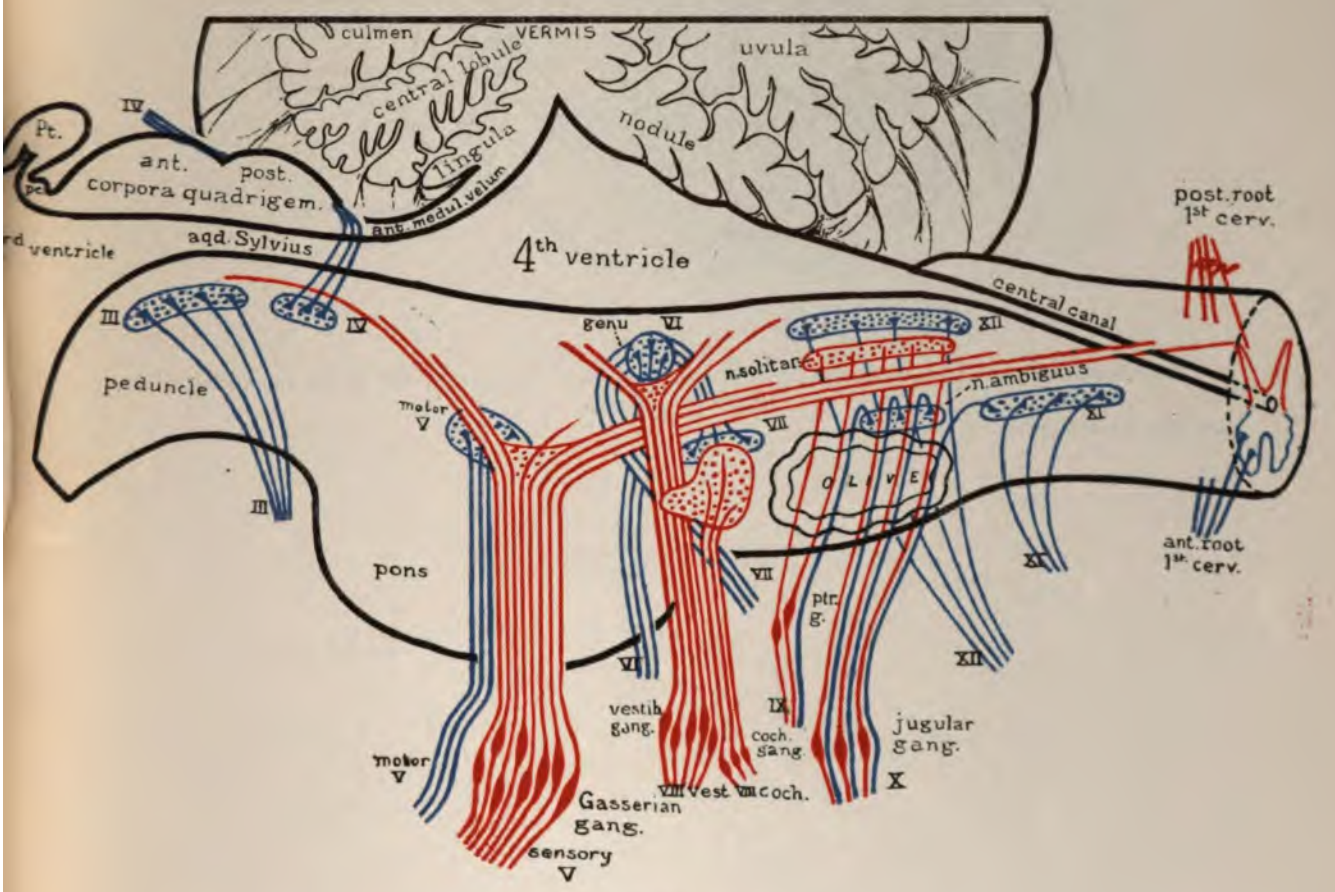


FIG. 19

Schematic representation of brain stem; showing nuclei and nerve roots. The sensory nuclei and nerve roots are colored red, the motor blue.

See 1321-4-39-48-66-87-90, 1408.

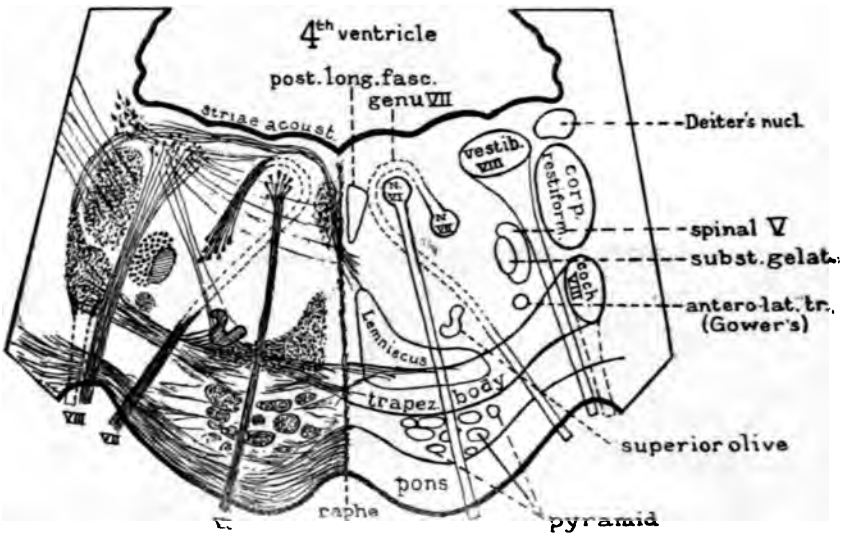


FIG. 20

Diagrammatic transverse section through the pons at a level slightly posterior to the superficial origin of the trigeminus.

See 1292, 1321-4-43-7-66-95, 1401-8.

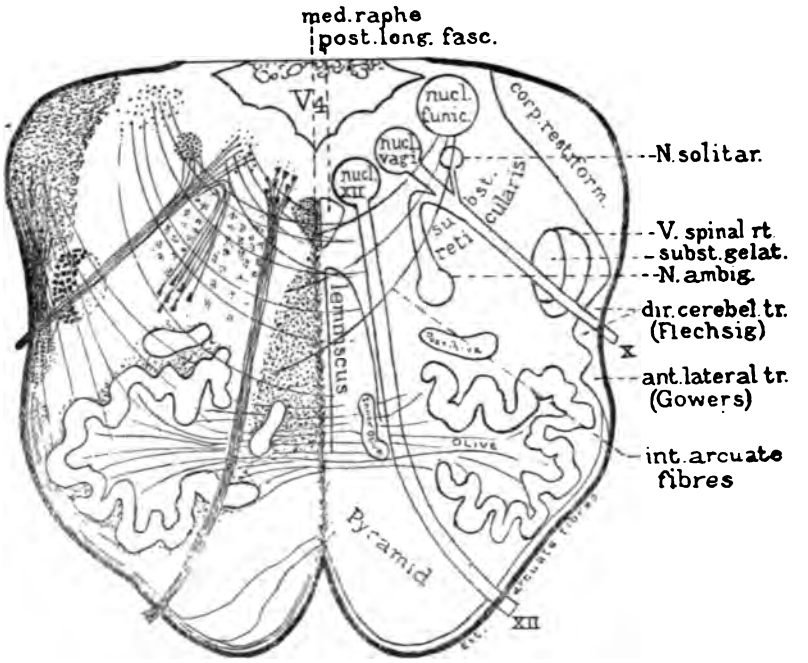


FIG. 21

Diagrammatic transverse section through the medulla, approximately near its middle.

See 1290-1, 1321-4-94, 1401-8.

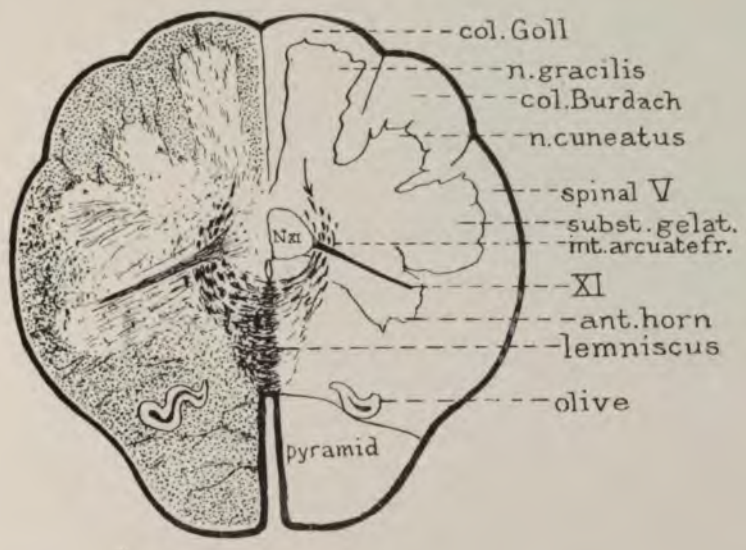


FIG. 22

Transverse section of medulla just above motor decussation and just above line of junction with the cord showing the sensory decussation and the topography of the lowest level of the medulla.

See 1290-1.

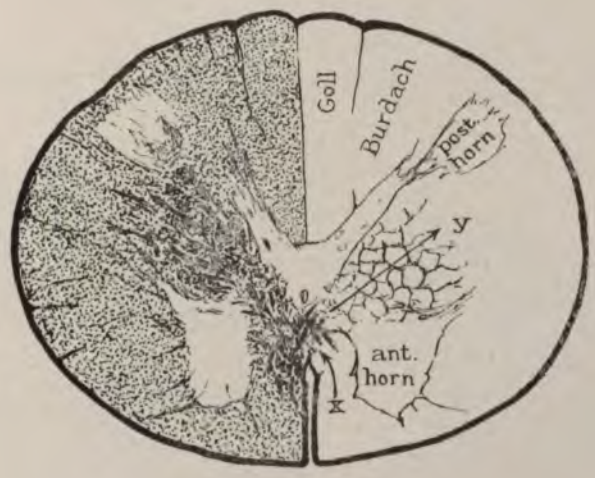


FIG. 23

Transverse section of the cord just at the line of junction with the medulla, showing the motor decussation and the topography of the uppermost level of the cord.

See 1290-1.

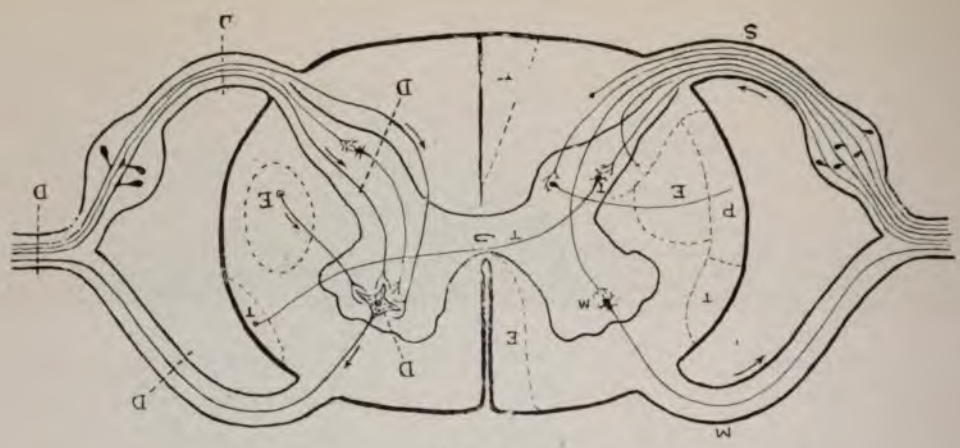


FIG. 24

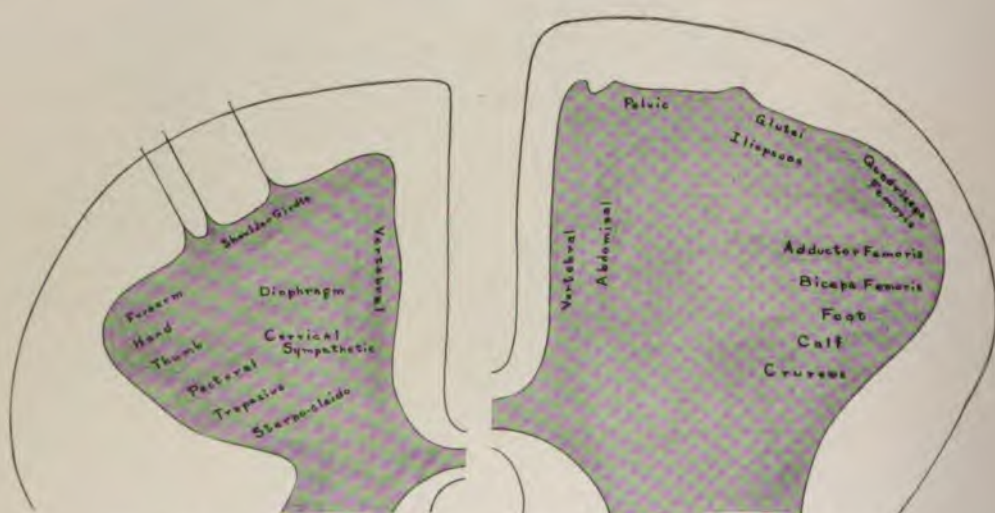
DIAGRAMMATIC SECTION OF THE SPINAL CORD TO ILLUSTRATE ITS PHYSIOLOGY

Left side shows situation of lesions causing disorders of motion and sensation.

Right side shows situation of lesions causing disorders of reflex activity.

Destructive lesions at M or E cause diminution, slight irritative lesions, exaggeration, of motion. Destructive lesions at S cause permanent anesthesia, analgesia, thermic anesthesia and loss of muscle sense. Destructive lesions at T cause analgesia and thermic anesthesia. Destructive lesions at P cause ataxia. Destructive lesions at K cause loss of muscle sense, ataxia and anesthesia. Irritative lesions at S, K, T, or P, may cause exaggeration, or perversion, or both, of sensation. Destructive lesions at D cause diminution, and at E, exaggeration, of reflex activity. Slight irritative lesions at D cause exaggeration, and at E diminution, of reflex activity.

Symptoms of lesions at M are described in 252, 263, 495, 547-8, 791, 1148-9, 1233 and 1324; at E in 251, 254, 256, 525-6, 799-800, 1212 and 1384-6-9; at S in 826; at T in 1369-71-3; at P in 281, 642-53; at K in 280, 654, 786, 1322-60-3-4 and 1406. The results of lesions at D and E are discussed in Chart Va.



Cervical Enlargement

Lumbar Enlargement

FIG. 25

LOCALIZATION OF NUCLEI IN THE ANTERIOR HORNS OF THE SPINAL CORD
(After Edinger modified from Sano.)

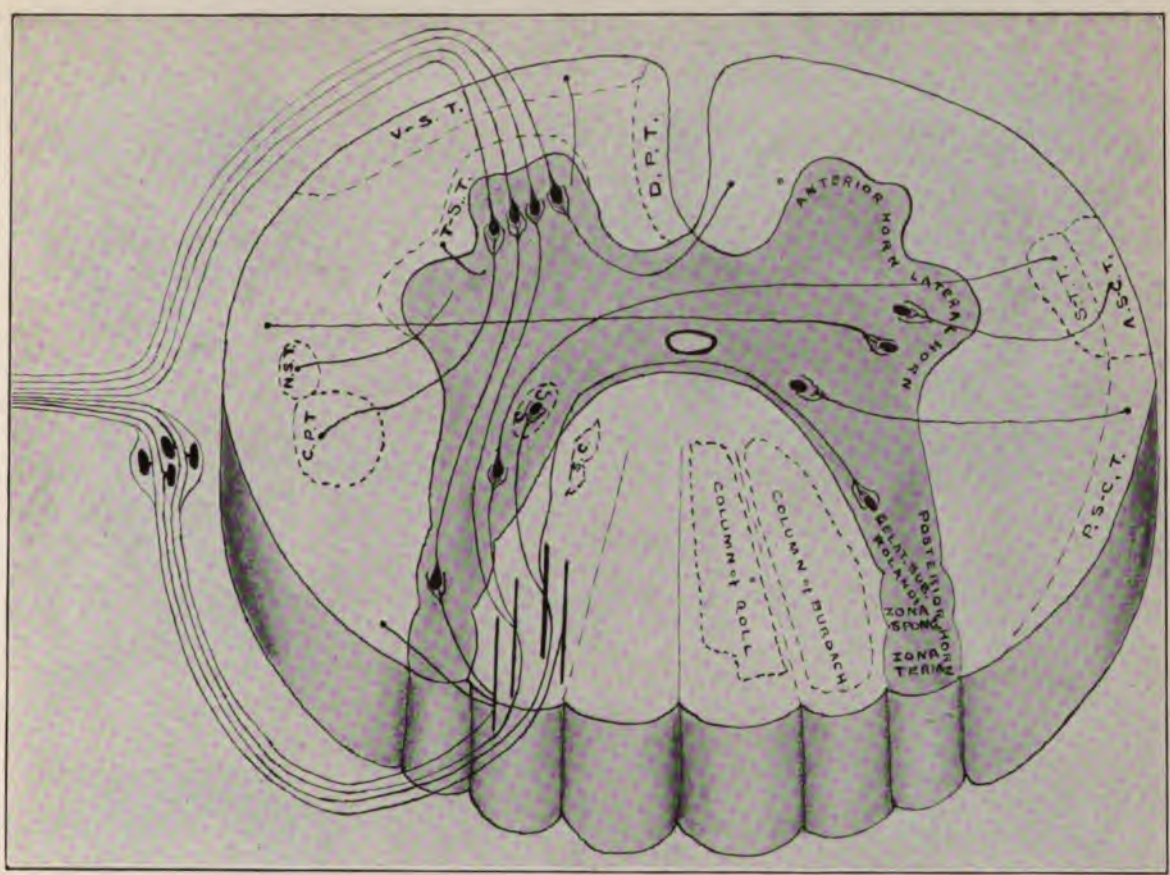


FIG. 26

A SCHEMATIC REPRESENTATION OF A TRANSVERSE SECTION OF THE SPINAL CORD, SEVERAL LEVELS BEING COMBINED INTO ONE

DESCENDING TRACTS

V. S. T. = vestibulo-spinal tract
T. S. T. = tecto-spinal tract
D. P. T. = direct pyramidal tract } cortico-spinal
C. P. T. = crossed pyramidal tract } tract
N. S. T. = rubro-spinal and thalamo-spinal tracts
S. C. = Schultze's comma

ASCENDING TRACTS

S. T. T. = spino-thalamic tract } (Gowers' tract)
A. S. C. T. = anterior spino-cerebellar tract } (Gowers' tract)
P. S. C. T. = posterior spino-cerebellar tract (Flechsig's tract)
C. C. = Clark's column

On the left side of the cord are represented the nerve roots and those bundles of long fibers in the white columns which carry impulses downward from the brain to the spinal cord, and on the right side are represented those bundles of long fibers in the white columns which carry impulses upward from the spinal cord or spinal ganglia to the brain. It hardly needs to be stated that, although in this figure these long bundles of fibers are represented on one side only, they are really situated symmetrically on each side of the cord. The short fibers which connect different levels of the cord together are not represented in the figure.

Lesions involving the pyramidal tract give rise to a spastic paralysis described under 251, 254, 525-6, 547, 660, 799-800, 1212 and 1384-6-9. Lesions involving the anterior horns give rise to atrophic paralysis, the acute forms of which are described under 495, 791, 1148, 1233 and 1324; while the chronic forms are described under 547-8, 695, 1150 and 1324. Lesions involving the posterior horn give rise to symptoms described under 1322. Lesions of posterior columns give rise to symptoms described under 785, 1302, 1347, 1350-1 and 1396. Lesions of the spino-cerebellar tract give rise to symptoms described under 281 and 653. Lesions of the spino-thalamic tract and of the anterior commissure of the gray matter give rise to symptoms described under 365, 812 and 1354. Lesions of the whole of one lateral half of the cord give rise to symptoms described under 442, 509, 840, 981 and 1405; while lesions of the whole transverse section of the cord give rise to symptoms described under 485, 513-4-17-8, 520-1-50-3, 793-8, 827-30-1-8, 980, 1149, 1329-30 and 1404-7. Lesions of posterior spinal ganglion give rise to symptoms described under 940-78.

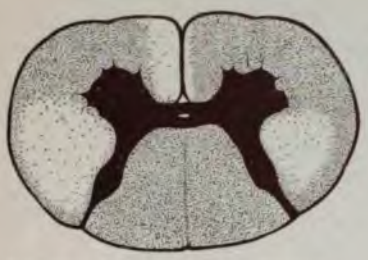
FIG. 27

Schematic representation of the more important diseases of the spinal cord.

Locomotor Ataxia
(lumbar region)

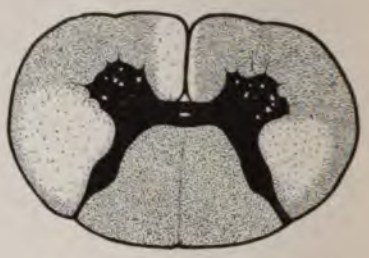
See 248, 345, 409-12-3-23, 661, 759, 785, 829, 891, 894, 912, 979, 988, 1004, 1015, 1172, 1186, 1217 and 1231

Locomotor Ataxia
(cervical region)

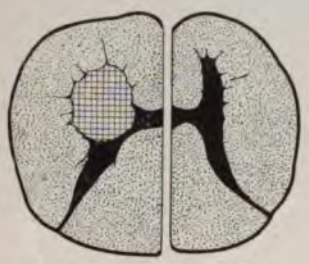


Amyotrophic Lateral Sclerosis

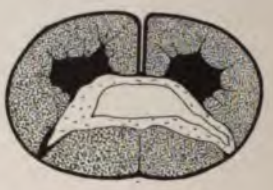
See 525, 547-8, 670, 695, 800 and 1150



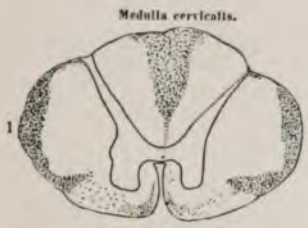
Descending Degeneration of
Pyramidal Tracts

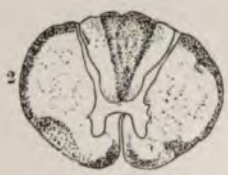


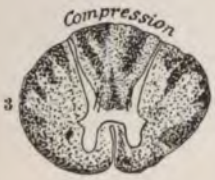
Acute Stage Chronic Stage
Anterior Poliomyelitis
See 695, 791, 800, 1148, 1150
and 1233



Syringomyelia
See 693, 802-40-2, 1009, 1152-70-87
and 1370-2







Compression Myelitis with the consequent Ascending and Descending Degenerations. See 520, 798.

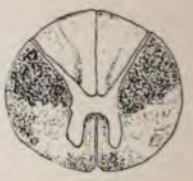
No 3 shows the point of the compression with the whole transverse section of the cord the seat of an inflammation.

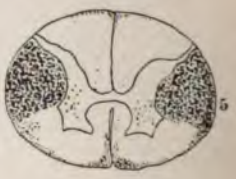
No. 1 shows ascending degeneration of the columns of Goll, of the spino-thalamic tracts, and of the anterior and posterior spino-cerebellar tracts.

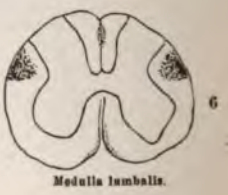
No. 2, close to the lesion, shows in addition a slight degeneration of the columns of Burdach.

Nos. 4-6 show degeneration of the crossed and direct pyramidal tracts, of the vestibulo-spinal, rubro-spinal, and thalamo-spinal tracts and of Schultze's comma.

The upper series faces up and the lower down.







SCHEMATIC REPRESENTATION OF SOME POINTS IN THE PHYSIOLOGY AND PATHOLOGY OF THE SPINAL CORD AND PERIPHERAL NERVES

FIG. 28. Diagram to illustrate the mechanism of the bladder reflex.

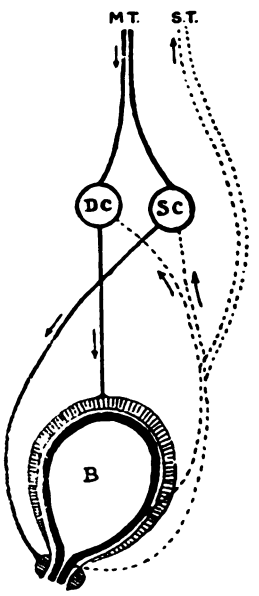
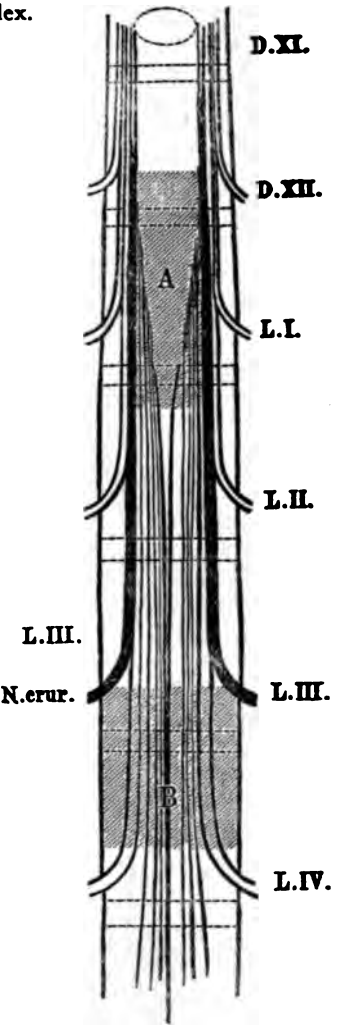


FIG. 28

B represents the bladder. S C represents the reflex centre, with its motor and sensory neurons, for the sphincter of the bladder, which is excited to action by urine in the neck of the bladder or in the prostatic urethra. DC represents the reflex centre, with its motor and sensory neurons, for the detrusor of the bladder, which is excited to action by the distention of the walls of the bladder. These two reflexes are antagonistic and the sensory surface irritated being much larger in the latter (D C), than in the former (S C), reflex, the detrusor reflex will eventually overpower the sphincter reflex under normal conditions. S T represents the sensory tract connecting the bladder with the brain, by means of which the individual is informed as to the degree of fulness of the bladder. M T represents the motor tract conecting the cerebral with the spinal centre by means of which the individual can inhibit the activity of either centre (up to a certain degree) and increase the activity of the antagonistic centre.

Fig. 29 illustrates effects of lesions of cauda equina.



(After Fr. Schultze-Köster.

FIG. 29

If the lesion is at "A" there is complete motor paralysis of both legs, and complete anesthesia of thewhole of both legs and of the perineum, buttocks, scrotum and penis, and all reflexes of the legs are abolished.

If the lesion is at "B" there is complete motor paralysis of both legs, except the flexors of the thigh and the extensors of the leg, and complete anesthesia of the perineum, buttocks, scrotum and penis, and of the posterior surface of the thighs, the posterior and lateral surfaces of the legs, and all of the foot, except a small area on its inner surface. All the reflexes of the legs except the knee-jerks are abolished.

In both cases the muscles atrophy, there is no zone of hyperesthesia above the anesthesia and the bladder and rectum show a motor and sensory paralysis.

If the lesion is limited to the conus medullaris there is a paralysis of the rectum and bladder and an anesthesia of the penis, scrotum, perineum, one inch about anus, and the upper two-thirds of the posterior surface of the thighs. Otherwise there is no paralysis of motion or sensation.

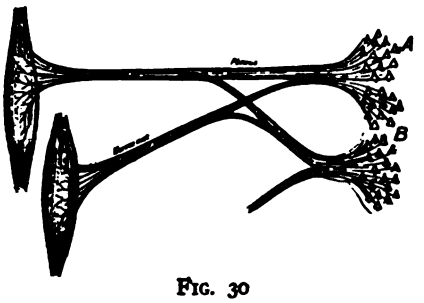


FIG. 30

Showing the innervation of muscles through more than one nerve root, so that the destruction of one nerve root or of one group of nerve cells does not cause a complete and permanent paralysis.

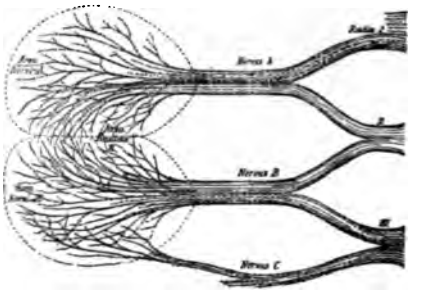


FIG. 31

A diagram showing that a given sensation area of the skin is supplied by filaments from several nerve roots; so that division of one root does not necessarily produce total anesthesia. It also shows the peripheral overlapping; so that the area supplied by one nerve can be almost completely supplied by neighboring nerves.

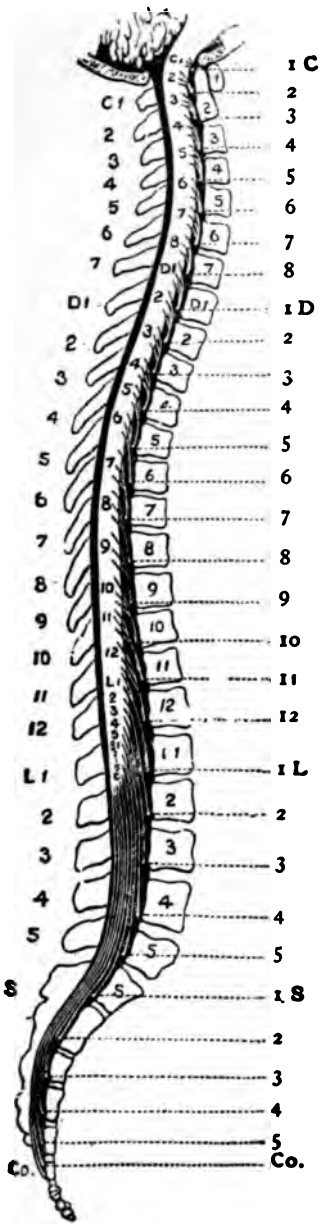


FIG. 32.

MOTOR AND REFLEX FUNCTIONS OF THE SPINAL-CORD SEGMENTS (MODIFIED AFTER STARR AND EDINGER)

SEGMENT	MUSCLES	REFLEXES
Cervical 2-3	Sternomastoid	
	Trapezius	
	Scaleni	
	Small rotators of head	
	Diaphragm	
	Lev. ang. scap.	Dilatation of pupil by irritating side of neck, 4 cervical to 1 dorsal
	Rhomboids	
4	Spinati	
	Deltoid	
	Supinat. long	Scapular reflexes, 5 C-1 D
5	Biceps	Supinat. long., 5 C
	Supinat. brev.	
	Serrat. mag.	
	Pectoralis (clav.)	Biceps, 5-6 C
	Teres minor	Triceps, 6 C
6	Pronators	Posterior wrist, 6-8 C
	Brachialis ant.	Scapulo-humeral, 7 C
	Triceps	
7	Long extensors of wrist and fingers	Anterior wrist, 7-8 C
	Pectoralis (costal)	
	Latiss. dorsi	Palmar, 7 C-1 D
	Teres maj.	
8	Long flexors, wrist and fingers	Epigastric, 4-7 D
Dorsal 1	Extensors of thumb	
1	Intrinsic hand-muscles	Abdominal, 7-11 D
2-12	Dorsal and abdominal muscles	
Lumbar	Abdominal muscles	Cremaster, 1-3 L
1	Iliacus	
	Psoas	Patellar, 2-4 L
2	Sartorius	Bladder, 2-4 L
	Flexors of knee	
	Quad. femoris	
3	Int. rotators of thigh	
	Adductors of thigh	Rectal, 4 L-2 S
4	Abductors of thigh	
	Tibialis ant.	Gluteal, 4-5 L
	Calf-muscles	
5	Ex. rotators of thigh	
	Extensors of toes	Ankle-clonus, Achilles, } 1-3 S
Sacral 1-2	Peronei	
	Long flex. of toes	Plantar, 1-2 S
	Intrinsic foot-muscles	Anal, Virile, } 3-5 S
3-5	Perineal muscles	

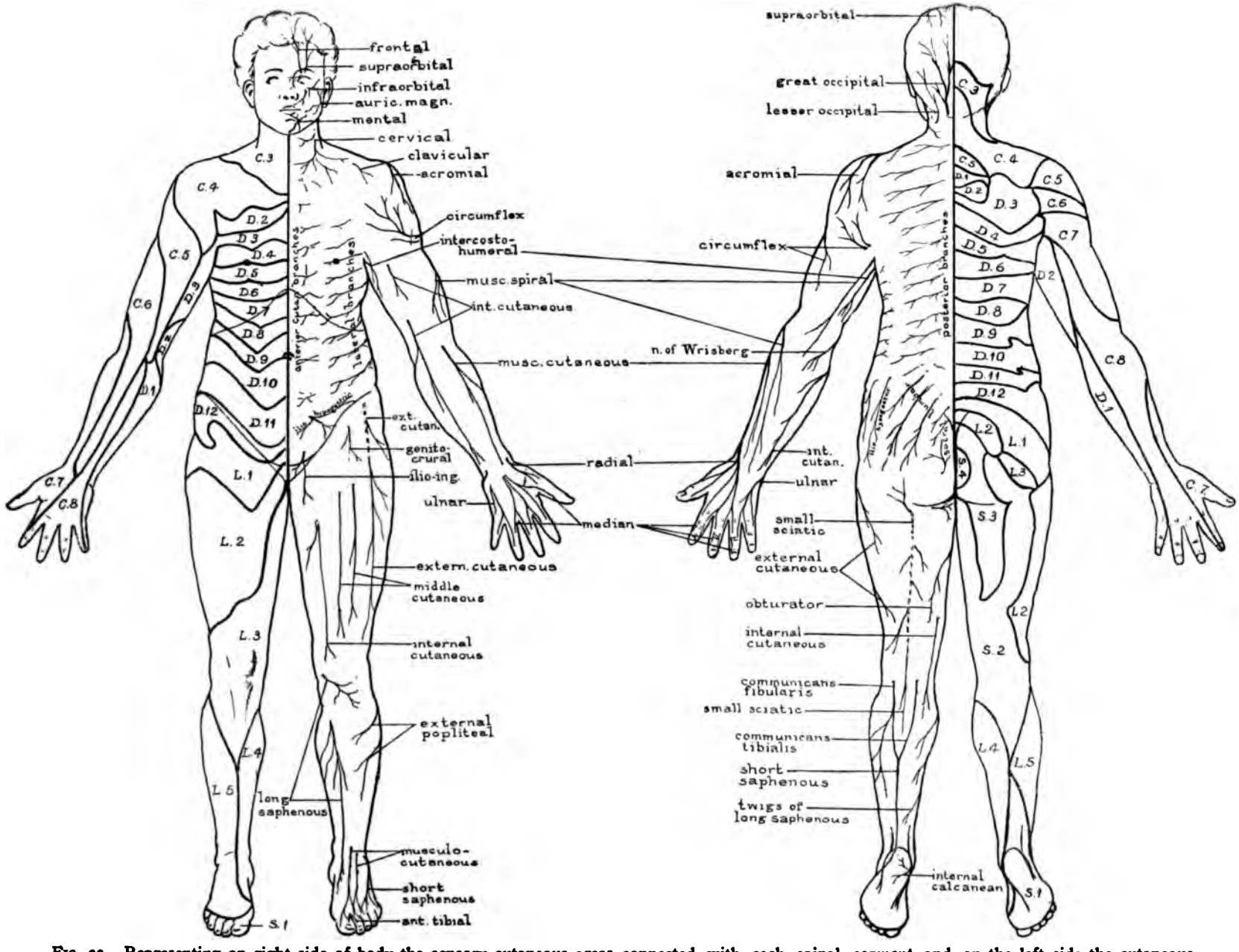


FIG. 33.—Representing on right side of body the sensory cutaneous areas connected with each spinal *segment* and on the left side the cutaneous distribution of the sensory *nerves*. See 636, 700-21, 824-6 and 1321-4.

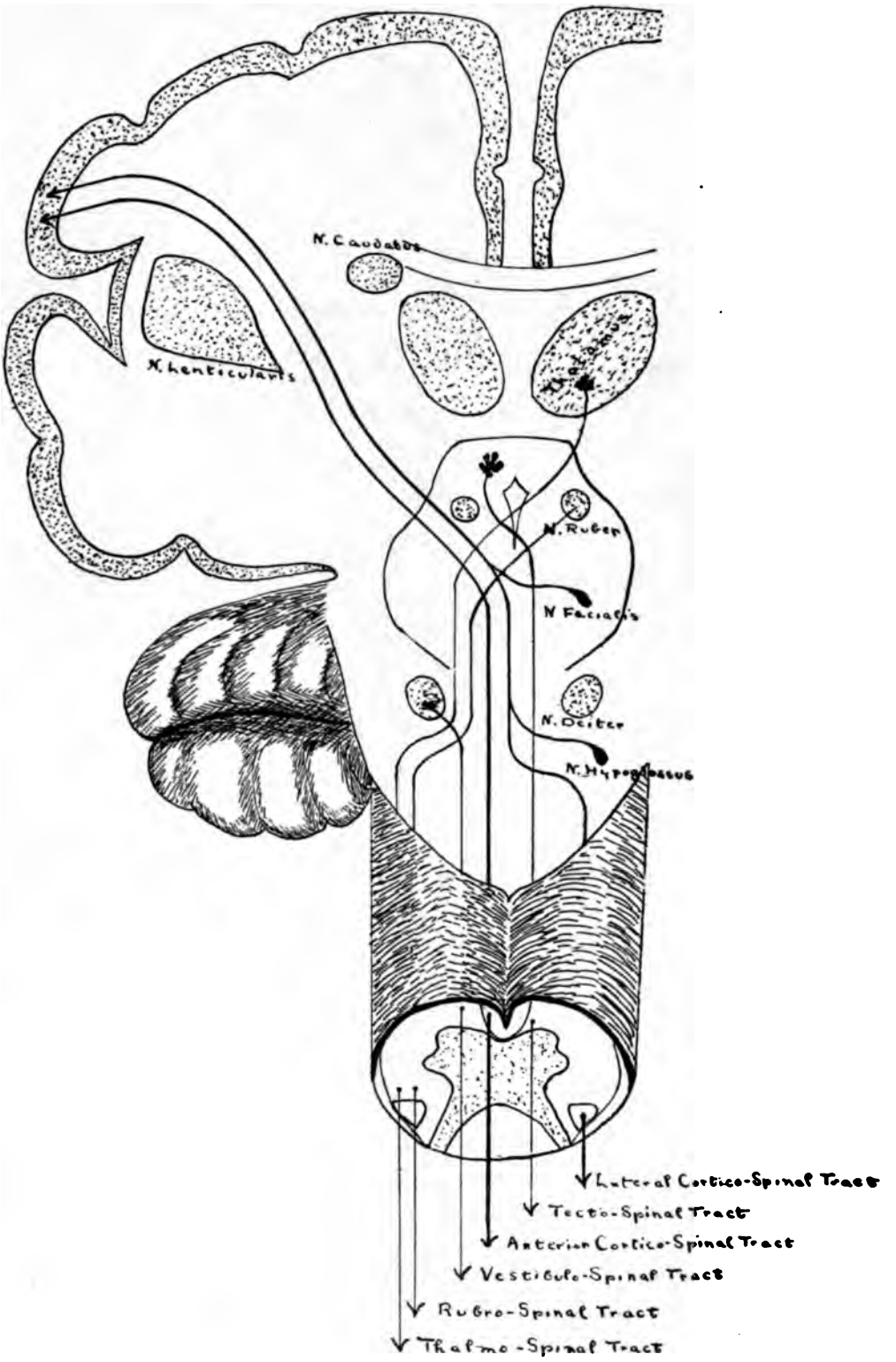


FIG. 34

LONG MOTOR PROJECTION TRACTS

For lesions involving these tracts see under Fig. 26

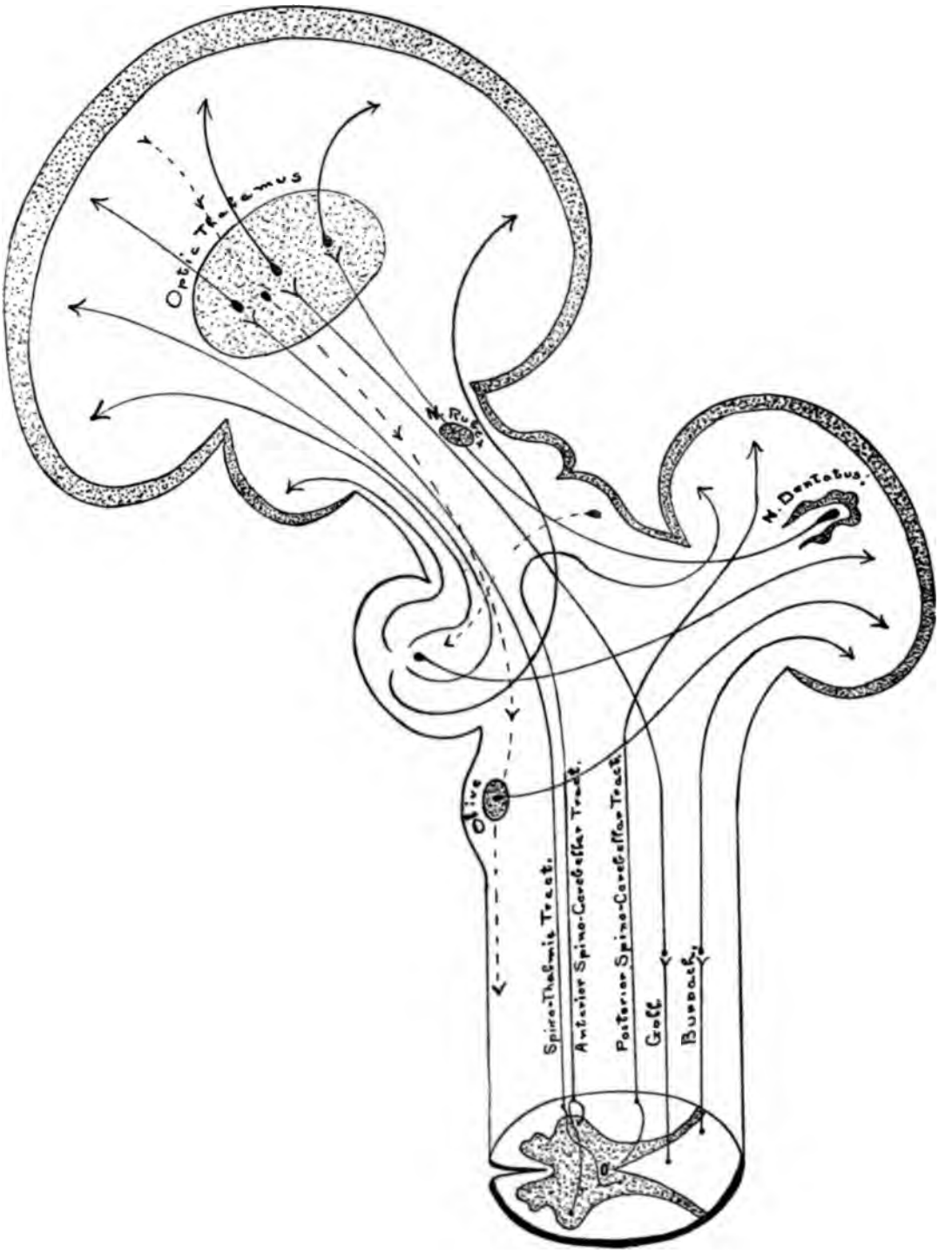


Fig. 35

LONG SENSORY PROJECTION TRACTS

For lesions involving these tracts see under Fig. 26

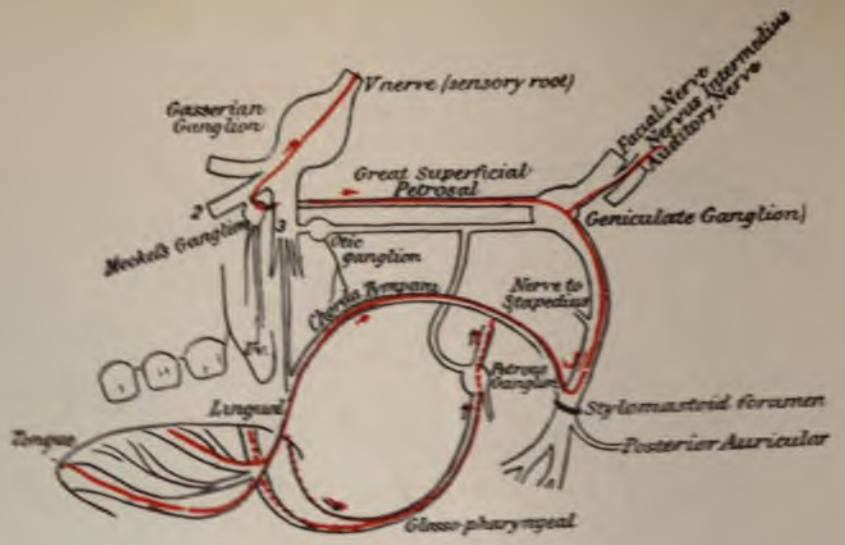


FIG. 36

DIAGRAM OF TRIGEMINAL, FACIAL AND GLOSSO-PHARYNGEAL NERVES, SHOWING COURSE OF TASTE FIBRES.

(After Purves Stewart)

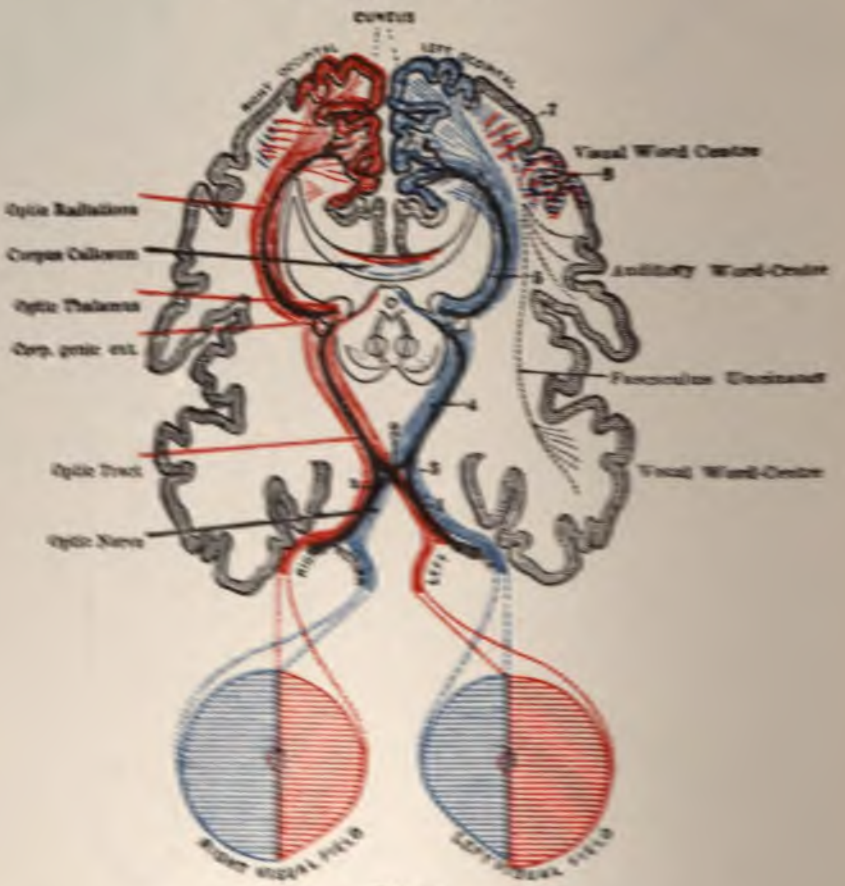


FIG. 37

DIAGRAM ILLUSTRATING HEMIANOPIA

(Modified from Violet)

Lesion at 1 produces blindness of one eye.
Lesion at 2 produces bi-temporal hemianopia.
Lesion at 3 produces bi-nasal hemianopia.
Lesion at 4 produces R. hemianopia with hemiopic pupil reaction.
Lesion at 5 produces R. hemianopia with normal pupil reaction.
Lesion at 6 produces R. hemianopia with normal pupil reaction.
Lesion at 7 produces psychic blindness.
Lesion at 8 produces Alexia.

The heavy black lines represent the fibers from the macula lutea in each retina, the point of most or clearest vision.

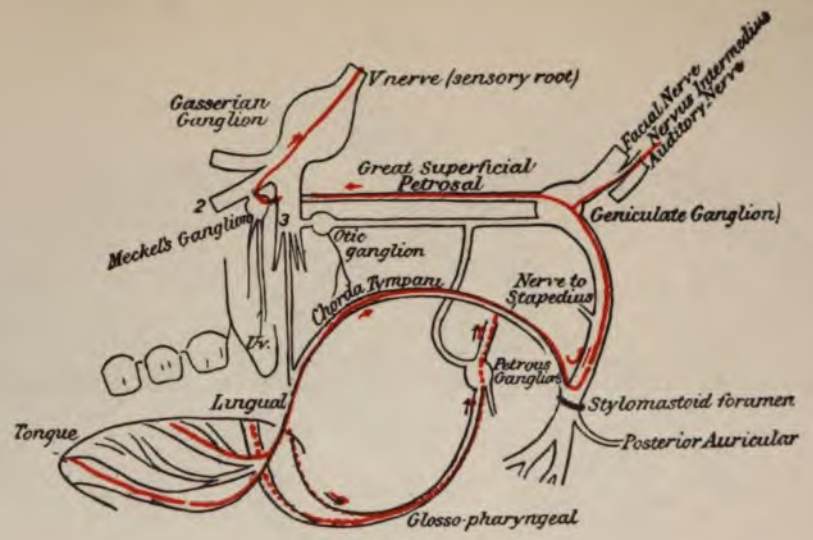


FIG. 36

DIAGRAM OF TRIGEMINAL, FACIAL AND GLOSSO-PHARYNGEAL NERVES, SHOWING COURSE OF TASTE FIBRES.

(After Purves Stewart)

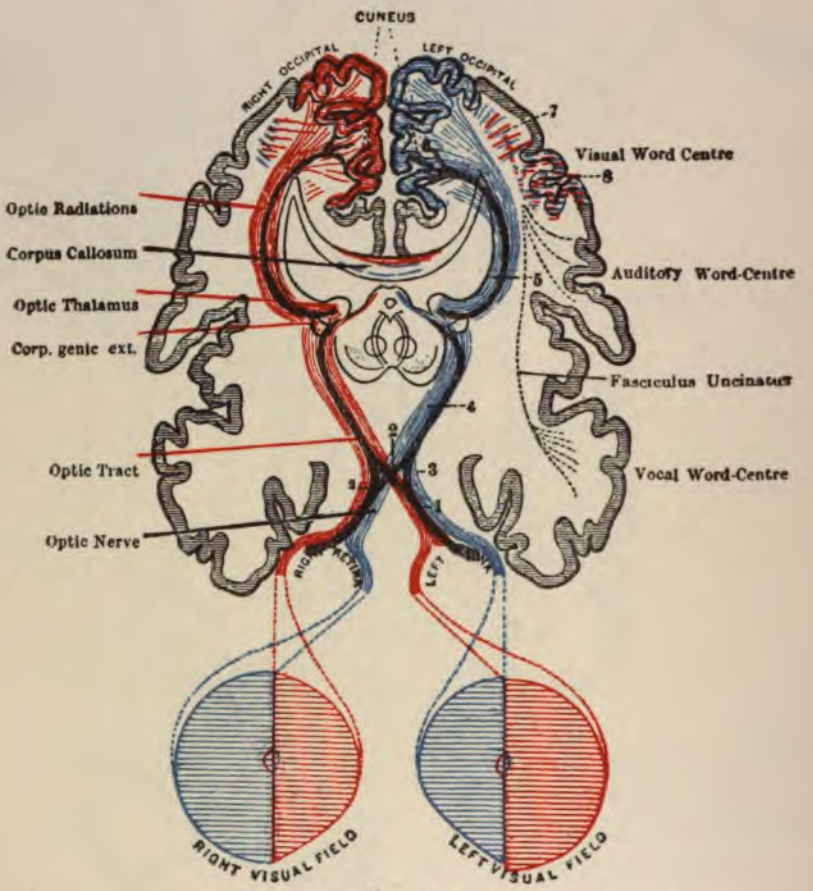


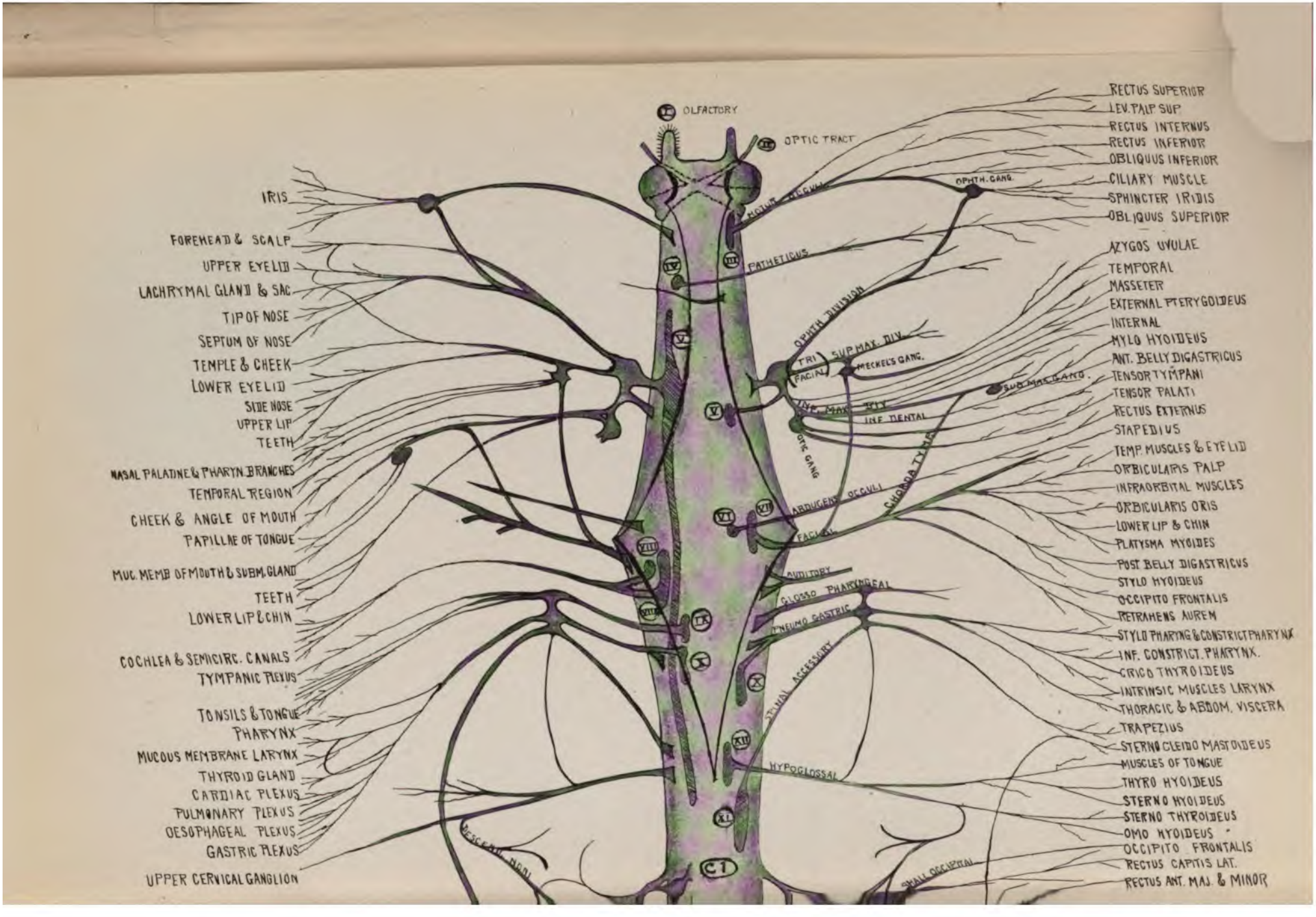
FIG. 37

DIAGRAM ILLUSTRATING HEMIANOPIA

(Modified from Vialet)

Lesion at 1 produces blindness of one eye.
Lesion at 2 produces bi-temporal hemianopia.
Lesion at 3 produces bi-nasal hemianopia.
Lesion at 4 produces R. hemianopia with hemiopic pupil reaction.
Lesion at 5 produces R. hemianopia with normal pup reaction.
Lesion at 6 produces R. hemianopia with normal pup reaction.
Lesion at 7 produces psychic blindness.
Lesion at 8 produces Alexia.

The heavy black lines represent the fibers from the macula lutea in each retina, the point of central or clearest vision.


OLFACTORY
OPTIC TRACT
OPHTH. GANG.
RECTUS SUPERIOR
LEV. PALP. SUP.
RECTUS INTERNUS
RECTUS INFERIOR
OBLIQUUS INFERIOR
CILIARY MUSCLE
SPHINCTER IRIDIS
OBLIQUUS SUPERIOR
IRIS
FOREHEAD & SCALP
UPPER EYELID
LACHRYMAL GLAND & SAC
TIP OF NOSE
SEPTUM OF NOSE
TEMPLE & CHEEK
LOWER EYELID
SIDE NOSE
UPPER LIP
TEETH
NASAL PALATINE & PHARYN. BRANCHES
TEMPORAL REGION
CHEEK & ANGLE OF MOUTH
PAPILLAE OF TONGUE
MUC. MEMB. OF MOUTH & SUBM. GLAND
TEETH
LOWER LIP & CHIN
COCHLEA & SEMICIRC. CANALS
TYMPANIC PLEXUS
TONSILS & TONGUE
PHARYNX
MUCOUS MEMBRANE LARYNX
THYROID GLAND
CARDIAC PLEXUS
PULMONARY PLEXUS
OESOPHAGEAL PLEXUS
GASTRIC PLEXUS
UPPER CERVICAL GANGLION
PATHETICUS
OPHTH. DIVISION
TRI
FACIAL
SUP. MAX. DIV.
MECKEL'S GANG.
SUB. MAX. GANG.
INF. MAX. DIV.
INF. DENTAL
OTIC GANG
CHORDA TYMPANI
ABDUCENS OCULI
FACIAL
AUDITORY
GLOSSO PHARYNGEAL
PNEUMO GASTRIC
SPINAL ACCESSORY
HYPOGLOSSAL
SMALL OCCIPITAL
AZYGOS UVULAE
TEMPORAL
MASSETER
EXTERNAL PTERYGOIDEUS
INTERNAL
MYLO HYOIDEUS
ANT. BELLY DIGASTRICUS
TENSOR TYMPANI
TENSOR PALATI
RECTUS EXTERNUS
STAPEDIUS
TEMP. MUSCLES & EYELID
ORBICULARIS PALP.
INFRAORBITAL MUSCLES
ORBICULARIS ORIS
LOWER LIP & CHIN
PLATYSMA MYOIDES
POST. BELLY DIGASTRICUS
STYLO HYOIDEUS
OCCIPITO FRONTALIS
RETRAHENS AUREM
STYLO PHARYNG & CONSTRICT PHARYNX
INF. CONSTRICT. PHARYNX.
CRICO THYROIDEUS
INTRINSIC MUSCLES LARYNX
THORACIC & ABDOM. VISCERA
TRAPEZIUS
STERNO CLEIDO MASTOIDEUS
MUSCLES OF TONGUE
THYRO HYOIDEUS
STERNO HYOIDEUS
STERNO THYROIDEUS
OMO HYOIDEUS
OCCIPITO FRONTALIS
RECTUS CAPITIS LAT.
RECTUS ANT. MAJ. & MINOR
C1

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Roman numerals indicate charts. Arabic numerals indicate marginal numbers. Arabic numerals preceded by "p" indicate pages.